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A POSSIBLE MENACE

It is not generally appreciated among the profession in this country that many of our veterinary practice acts are lamentably weak in defining the eligibility requirements for an applicant for a license to practice veterinary medicine. From inquiries we have made recently, we find that many veterinarians are of the opinion that graduation from a recognized veterinary college is a minimum requirement in all states. Unfortunately, this is not the case, as at least sixteen veterinary practice acts in as many states make no such provision. Almost without exception, the laws in these states make it possible for anybody to take the examinations.

Now it is true that taking an examination is one thing and passing it is something else, but let us look at the records. In the state of Tennessee, five non-graduates were granted licenses during the past ten years. During the period since 1924, Oklahoma has issued 13 non-graduate licenses. Part of these were granted to graduates of non-recognized colleges. Six other states have licensed one non-graduate each during the past ten years.

From these facts it will be seen that there is not much to the argument that the examinations can be made so stiff that nobody but a graduate veterinarian can pass them. On the other hand, it may be that in some states the examinations are much more difficult than in others. At the recent meeting of the Oklahoma Board of Veterinary Medical Examiners, six non-graduates were examined and all failed. During the past ten years, five non-graduates have taken the examinations in Washington and failed, with averages ranging from 12.8 to 57 per cent.

It may be argued that the failure of a law to require that an applicant for a license be a graduate of a recognized college is a good thing. In at least one state this situation probably prevented the repeal of the veterinary practice act altogether. It was shown that the law did not prevent anybody from taking the examinations and this appeased the politicians—temporarily, at least.

But there is one rather disquieting phase of this situation. Among the veterinary practice acts that require a candidate for a license to practice to be a graduate of a recognized veterinary college, we have been able to find but one that requires the applicant to be a graduate of a four-year school, even though all of the existing veterinary colleges in the United States and Canada have been on a four-year basis for about 20 years. It is true that many of the veterinarians now active are either two-year or three-year men and it would be unfair to these men to pass laws that would render them ineligible to secure a license to practice in any state, but on the other hand, it would seem in order to have more of our veterinary practice acts in consonance with present-day veterinary education.

During recent years, hundreds of young men have knocked on the doors of veterinary colleges but have been refused admission. How long will a demand of this kind be allowed to go unsatisfied? It is almost axiomatic that when there is a real demand for something that is at present unobtainable, sooner or later somebody will arise to suply that demand. That might happen with veterinary education. And just suppose that somebody with a bright idea and some capital should decide that a four-year course is too long or not necessary and start a three-year veterinary college. Graduates would be eligible to take state board examinations in almost any state in the Union. Such a course of events is not pleasant to contemplate, but stranger things have happened.

EXECUTIVE BOARD ELECTIONS

The primary elections in Executive Board District 2 (Pennsylvania, New Jersey and Delaware) and District 3 (Indiana, Illinois and Wisconsin) had an unusual outcome in both cases. The voting was comparatively light and very much scattered. In District 2 there were only 36 ballots cast, but these contained the names of 14 nominees, while in District 3 the names of 15 nominees appeared on 156 ballots cast. When it came to tabulating the votes in District 2, it was found that first, second, third and fourth places were not in doubt, but there were ten nominees tied for fifth place with one vote apiece. Much the same situation was found in District 3, where twelve nominees were tied for fourth place, with one vote each.

About ten years ago a similar situation developed in one of the regular Executive Board elections and, with that as a precedent, a letter was written to each of the tied nominees in both districts, explaining the situation and asking each one whether he desired his name to appear on the ballot. In District 3, all of the nominees involved in the tie stated that they did not choose to run. In District 2, six nominees replied that they were not candidates, one stated that he was, and three failed to reply to our letter. One nominee was found to be ineligible. As a result of the withdrawals, only three nominees appeared on the ballot in District 3 and seven names constituted the ballot in District 2.

DISTRICT 2

ALTHOUSE, E. P. — Sunbury, Pa.

Practitioner. Graduate of University of Pennsylvania, 1903. Joined A. V. M. A., 1903. Member of Committee on Program, 1928-29; secretary of Section on General Practice, 1926-29; member of Executive Board (District 2), 1928-.

EMMERSON, M. A. — Philadelphia, Pa.

Assistant Professor of Veterinary Surgery and Obstetrics, University of Pennsylvania. Graduate of Iowa State College, 1925. Joined A. V. M. A., 1926. Secretary of Section on General Practice, 1937-.

HARDENBERGH, J. G. — Plainsboro, N. J.

Laboratory Director, Walker-Gordon Laboratory Company. Graduate of University of Pennsylvania, 1916. Joined A. V. M. A., 1918. Member of Special Committee on Distemper, 1926-27; resident secretary for New Jersey, 1929-33 and 1934-35; chairman of Special Committee on Food Hygiene, 1936-.

HENDERSHOTT, R. A. — Trenton, N. J.

Chief, New Jersey Bureau of Animal Industry. Graduate of Ohio State University, 1917. Joined A. V. M. A., 1919. Resident secretary for New Jersey, 1936-; member of Committee on Local Arrangements for New York Meeting, 1937-38.

IVENS, W. H. Philadelphia, Pa.

Practitioner. Graduate of University of Pennsylvania, 1910. Joined A. V. M. A., 1916. Chairman of Section on General Practice, 1932-35.

MORRIS, M. L. Stelton, N. J.

Practitioner. Graduate of Cornell University, 1926. Joined A. V. M. A., 1929.

PICKUP, E. C. Union City, Pa.

Practitioner. Graduate of Ontario Veterinary College, 1916. Joined A. V. M. A., 1917.

DISTRICT 3

AXBY, J. L. Indianapolis, Ind.

State Veterinarian of Indiana. Graduate of Chicago Veterinary College, 1903. Joined A. V. M. A., 1908. Member of Committee on Local Arrangements for Indianapolis Meeting, 1911-12; member of Committee on Legislation, 1926-31 and 1937-; secretary of Section on Sanitary Science and Food Hygiene, 1933-35; member of Committee on Program, 1933-35.

MERILLAT, L. A. Chicago, Ill.

Manager, Chicago Branch, Haver-Glover Laboratories. Graduate of Ontario Veterinary College, 1888. Joined A. V. M. A., 1893. Member of Committee on Resolutions, 1898-1900 and 1923-24; member (1899-1900, 1901-02, 1903-04) and chairman (1902-03) of Committee on Pharmacopoeia; member of Committee on Diseases, 1906-07; member of Executive Committee, 1906-07 and 1911-12; member of Committee on Intelligence and Education, 1910-11; member of Budget Committee, 1916-18 and 1924-25; member of Committee on Revision of Constitution and By-Laws, 1922-23; member of Committee on Policy, 1922-23 and 1924-25; resident secretary for Illinois, 1905-13 and 1915-16; secretary (1919-20) and chairman (1915-16) of Section on General Practice; secretary, 1916-17; second vice-president, 1906-07; president, 1924-25; member of Executive Board (District 3), 1928-.

WISNICKY, W. Madison, Wis.

Director of Wisconsin Department of Agriculture and Markets. Graduate of Kansas State College, 1926. Joined A. V. M. A., 1927. Member of Committee on Bang's Disease, 1930-; fifth vice-president, 1934-35.

APPLICATIONS FOR MEMBERSHIP

The number of applications for membership filed during the month of March totaled 26, which is very good as judged by previous records for the same month. During the period of 13 years during which the present method of handling applications has been in vogue, on only two occasions has March produced more applications than during the month just closed, in 1928 and again in 1936. The 26 applications which are given first listing this month are distributed among 14 states, with Minnesota again

leading the procession with five applications to her credit due to the splendid work of our resident secretary for the Gopher State, Dr. Joab P. Foster. Since the meeting in Omaha last summer, 134 applications have been filed and of this number 25 have been credited to Minnesota. California is in second place with eleven, Alabama is third with seven, Pennsylvania is fourth with six and Montana is fifth with five.

(See January, 1938 JOURNAL)

FIRST LISTING

CAIN, HAROLD W. 17422 Woodward Ave., Detroit, Mich.
V. S., B. V. Sc., Ontario Veterinary College, 1936
Vouchers: F. D. Egan and S. R. Elko.

CAMERON, JAMES A. Horse Infirmary, Union Stock Yards, Chicago, Ill.
M. D. V., McKillip Veterinary College, 1910
Vouchers: J. M. Handley and Israel Wallman.

COLLIER, JAMES R. 3441 Gurfeson Ave., Sioux City, Iowa
D. V. M., Ohio State University, 1932
Vouchers: Russel J. Stewart and T. L. Steenerson.

DEAL, BLAKELY T. 5350 Webster St., Philadelphia, Pa.
V. M. D., University of Pennsylvania, 1937
Vouchers: F. E. Lentz and M. A. Emmerson.

DUNN, WILLIAM T. Box 428, Hartsville, S. C.
B. S., University of Florida, 1932
D. V. M., Iowa State College, 1936
Vouchers: G. J. Lawhon and W. A. Barnette.

HAMNER, ROBERT H. 409 Post Office Building, Montgomery, Ala.
D. V. M., Alabama Polytechnic Institute, 1919
Vouchers: J. E. Greene and R. L. Mundhenk.

HINKLE, THOMAS C., JR. 732 N. 8th St., East Saint Louis, Ill.
B. S. A., Kansas State College, 1933
D. V. M., Kansas State College, 1936
Vouchers: Charles Barnes and R. W. Culbert.

KELLY, JAMES J. Marshall, Minn.
D. V. M., McKillip Veterinary College, 1917
Vouchers: Joab P. Foster and Leo S. Englerth.

KITCHEN, GEORGE R. Sumter, S. C.
D. V. M., Alabama Polytechnic Institute, 1929
Vouchers: J. E. Greene, R. L. Mundhenk and W. A. Barnette.

LAVITAN, MURRAY J. c/o City Health Dept., Charlotte, N. C.
D. V. M., Cornell University, 1937
Vouchers: H. D. Pritchett and A. A. Husman.

MCNEELEY, FRANK F. Colfax, La.
B. Sc., Louisiana State University, 1914
D. V. M., Kansas City Veterinary College, 1917
Vouchers: E. P. Flower and H. H. Baur.

MANTHEI, CHESTER A. 904 Acklen Ave., Nashville, Tenn.
D. V. M., Michigan State College, 1935
Vouchers: Hugh L. Fry and E. B. Parker.

MICHAEL, LLOYD J. 1805 Church St., Cleveland, Tenn.
D. V. M., Kansas State College, 1934
Vouchers: Hugh L. Fry and D. Coughlin.

MOIR, THOMAS A. 1249 Morningside Ave., Sioux City, Iowa
D. V. M., Colorado State College, 1927
Vouchers: I. E. Newson and Henry F. Winiecki.

MOLENGRAFT, JOSEPH A. 6317 S. Mozart St., Chicago, Ill.
D. V. M., Cincinnati Veterinary College, 1916
Vouchers: Israel Wallman and B. H. Steigleder.

NEIDIG, CHARLES C. Aitkin, Minn.
M. D. V., McKillip Veterinary College, 1909
Vouchers: Joab P. Foster and Leo S. Englerth.

RILEY, EMMETT T. 336 Post Office Building, Oklahoma City, Okla.
D. V. M., Texas A. & M. College, 1935
Vouchers: L. J. Allen and Wilbur McPherson.

RODE, WALTER F. White Bear, Minn.
D. V. M., McKillip Veterinary College, 1913
Vouchers: Joab P. Foster and George S. Failing.

SCHUBERT, CARL J. Slinger, Wis.
D V. M., Iowa State College, 1937
Vouchers: W. Wisnicky and James S. Healy.

STEPHENSON, HADLEY C. 105 Cornell St., Ithaca, N. Y.
B. S., Cornell University, 1919
D. V. M., Cornell University, 1920
Vouchers: H. J. Milks and J. N. Frost.

STEVENS, JOHN D. Box 852, Sequim, Wash.
B. S., D. V. M., State College of Washington, 1930
Vouchers: C. E. Sawyer and C. M. Hamilton.

SUTTON, KARL L. Morenci, Mich.
D. V. M., Michigan State College, 1936
Vouchers: Chas. Hodder and Wm. A. Rader.

TITTERUD, OSCAR H. Preston, Minn.
V. S., Ontario Veterinary College, 1905
M. D. C., Chicago Veterinary College, 1906
Vouchers: Joab P. Foster and Orval C. Selby.

VENZKE, WALTER G. Galva, Iowa
D. V. M., Iowa State College, 1935
M. S., University of Wisconsin, 1937
Vouchers: C. H. Covault and F. B. Hadley.

WENTWORTH, JAMES E. Rochester, Minn.
D. V. M., Kansas City Veterinary College, 1912
Vouchers: Joab P. Foster and Orval C. Selby.

YOUNG, ANDERSON V. Route 1, Box 167, Shreveport, La.
D. V. M., Texas A. & M. College, 1927
Vouchers: G. A. Bryson and H. H. Baur.

Applications Pending

SECOND LISTING

(See March, 1938, JOURNAL)

Becton, Lee M., 15 Virginia Ave., Staunton, Va.
Brock, Conway C., Lake City, Fla.
Creel, Ben W., c/o Board of Health, Mobile, Ala.
Eliason, Theodore R., 3632 Park Ave., Minneapolis, Minn.
Howell, William M., Alabama Polytechnic Institute, Auburn, Ala.
Ivens, William H., Jr., 60 Haverford Rd., Ardmore, Pa.
Neal, Allan J., 324 Essex St., Bangor, Me.
Pinckard, Wendell L., 312 W. Church St., Knoxville, Tenn.
Remer, Frederick B., Havre, Mont.

13th International Veterinary Congress

Zurich-Interlaken, Switzerland, August 20-27, 1938

APPLICATION FOR MEMBERSHIP

I desire to become a member of the 13th International Veterinary Congress, and enclose herewith a check (express or postal money order) for $7.00 in payment of the subscription as ordinary member. (Lady Members $1.25.)

Signature_____

Name (please print)_____

Address_____

_____

Official Position (if any)_____

_____

This form should be used only by veterinarians in the United States and U. S. possessions.

To be sent with remittance for the amount of the subscription to Dr. H. Preston Hoskins, Secretary
221 N. LaSalle St., Chicago, Ill.

Ritchie, Kenneth L., c/o County Agent's Office, Blackfoot, Idaho.
Russ, Robert S., 614 Post Office Building, Portland, Ore.
Starnes, Robert E., 817 W. Davis St., Dallas, Texas.
Strasburger, Russell F., Box 244, Newton, Conn.
Thames, Curtis B., Greenville, Ala.
Walker, Erling H., Auburn, Ala.
Watson, Lt. Edward J., Fort George Wright, Spokane, Wash.
Willard, Horace R., 1601 N. Ballinger St., Fort Worth, Texas.
Winters, Everett S., Alabama Polytechnic Institute, Auburn, Ala.

The amount which should accompany an application filed this month is $8.75, which covers membership fee and dues to January 1, 1939, including subscription to the JOURNAL.

COMING VETERINARY MEETINGS

Kansas City Veterinary Medical Association. Auditorium, General Hospital No. 1 Annex, Kansas City, Mo. April 18, 1938. Dr. S. J. Schilling, Secretary, Box 167, Kansas City, Mo.

San Diego County Veterinary Medical Association. Zoölogical Research Building, Balboa Park, San Diego, Calif. April 18, 1938. Dr. E. F. Sheffield, Secretary, 3895 Pacific Blvd., San Diego, Calif.

North-Central Iowa Veterinary Association. Fort Dodge, Iowa. April 20, 1938. Dr. H. C. Smith, Secretary, 2014 Eighth Ave. N., Fort Dodge, Iowa.

Southern California Veterinary Medical Association. Chamber of Commerce Building, Los Angeles, Calif. April 20, 1938. Dr. B. B. Coale, Secretary, 203 Administration Bldg., Union Stock Yards, Los Angeles, Calif.

Southwestern Minnesota Veterinary Medical Association. Saint James, Minn. April 26, 1938. Dr. Louis E. Stanton, Secretary, Jackson, Minn.

Keystone Veterinary Medical Association. School of Veterinary Medicine, 39th St. and Woodland Ave., Philadelphia, Pa. April 27, 1938. Dr. C. S. Rockwell, Secretary, 4927 Osage Ave., Philadelphia, Pa.

Massachusetts Veterinary Association. Hotel Westminster, Boston, Mass. April 27, 1938. Dr. H. W. Jakeman, Secretary, 44 Bromfield St., Boston, Mass.

Southern California, Veterinary Hospital Association of. Los Angeles, Calif. May 3, 1938. Dr. L. B. Wolcott, Secretary, 1434 W. Slauson Ave., Los Angeles, Calif.

Connecticut Veterinary Medical Association. Dr. George E. Bitgood's Hospital, Middletown, Conn. May 4, 1938. Dr. Geo. E. Corwin, Secretary, State Office Bldg., Hartford, Conn.

New York City, Veterinary Medical Association of. Hotel New Yorker, 8th Ave. and 34th St., New York, N. Y. May 4, 1938. Dr. J. B. Engle, Secretary, Box 432, Summit, N. J.

Saint Louis District Veterinary Medical Association. Melbourne Hotel, Saint Louis, Mo. May 4, 1938. Dr. Milton R. Fisher, Secretary, 3678 Dover Pl., Saint Louis, Mo.

Dallas-Fort Worth Veterinary Medical Society. Adolphus Hotel, Dallas, Texas. May 5, 1938. Dr. H. V. Cardona, Secretary, 2736 Purington Ave., Fort Worth, Texas.

Houston Veterinary Association. Houston, Texas. May 5, 1938. Dr. E. G. Pigman, Secretary, 4206 Polk Ave., Houston, Texas.

Ak-Sar-Ben Veterinary Medical Association. Fontenelle Hotel, Omaha, Neb. May 9, 1938. Dr. J. D. Ray, Secretary, 1124 Harney St., Omaha, Neb.

Chicago Veterinary Medical Association. Hotel Sherman, Chicago, Ill. May 10, 1938. Dr. O. Norling-Christensen, Secretary, Box 12, Wilmette, Ill.

Hudson Valley Veterinary Medical Society. Catskill, N. Y. May 11, 1938. Dr. J. G. Wills, Secretary, Box 751, Albany, N. Y.

Southeastern Michigan Veterinary Medical Association. Medical Arts Building, 3919 John R. St., Detroit, Mich. May 11, 1938. Dr. F. D. Egan, Secretary, 17422 Woodward Ave., Detroit, Mich.

Willamette Valley Veterinary Medical Association. Tillamook, Ore. May 14, 1938. Dr. Elwyn W. Coon, Secretary, Forest Grove, Ore.

Mississippi Valley Veterinary Medical Association. Galesburg Horse and Mule Barn, North Cherry St., Galesburg, Ill. May 26, 1938. Dr. Lyle A. Gray, Secretary, Bushnell, Ill.

Midwest Veterinary Conference and Clinic. Municipal Auditorium, Kansas City, Mo. June 7-9, 1938. Dr. S. J. Schilling, Corresponding Secretary, Box 167, Kansas City, Mo.

Ohio State University Veterinary Conference. Ohio State University, Columbus, Ohio. June 15-16-17, 1938. Dr. Oscar V. Brumley, Dean, Ohio State University, Columbus, Ohio.

American Veterinary Medical Association. Hotel Pennsylvania, New York, N. Y. July 5-9, 1938. Dr. H. Preston Hoskins, Secretary, 221 N. La Salle St., Chicago, Ill.

URINARY CALCULI WITH SPECIAL REFERENCE TO CATTLE AND SHEEP*

By I. E. NEWSOM, *Fort Collins, Colo.*
Colorado Agricultural Experiment Station

OUTBREAKS IN SHEEP

Our attention was first called to losses from urinary calculi in sheep near Fort Morgan, Colorado, in 1923. In that outbreak[1] some 25 lambs were lost out of 4,200 on feed. They had been in the feed-lots for five months before trouble was experienced. The ration was alfalfa, corn and linseed meal. Chemical analysis showed the calculi to be composed entirely of phosphates.

Sometime after this, sheep on beet-tops near Eaton were dying in considerable numbers from ruptured bladders. The loss in this band also was heavy, running to about 40 head before the sheep were ready for market.

At the Akron dryland experiment station, hardly a year passes without some loss in the small flock of sheep maintained there. In 1933, seven lambs died there out of 240 on feed, following a freeze-up of the water supply. The feed was largely composed of corn, cottonseed cake, cane, millet and bean straw.

Some years ago, a man near Brush lost heavily when feeding wheat. He changed his ration to corn and alfalfa and lost no more.

Near Yoder, Wyoming, out of 2,200 lambs on feed during this past winter, there were ten showing symptoms of calculi at one time. They were on alfalfa, corn, bran, dried pulp and cottoncake.

A man near Fort Collins has had an interesting experience. He has been feeding lambs for 17 years, with only an occasional case of what he called "water belly," until three years ago. Up to that time, he had fed alfalfa but since then he has used cane, oat and wheat straw, and corn fodder as roughage. Three years ago, he had a heavy loss; last year, he lost 5 per cent, and this year about 4 per cent from ruptured bladder. This year, he fed pea, oat and cane hay for roughage, and corn, cottoncake and beet syrup for concentrate. Last year, the feed was similar except that beet tops were included. He was also feeding a commercial mineral. When he changed to corn and alfalfa, the disease stopped and he had no further trouble.

*Presented at the seventy-fourth annual meeting of the American Veterinary Medical Association, Omaha, Neb., August 16-20, 1937.

These do not exhaust the outbreaks that have been called to our attention but they are the ones that have been most impressive. The belief is strong that the disease is either more prevalent or more frequently diagnosed in recent years.

Outbreaks in Cattle

Throughout eastern Colorado, the disease is so common in the winter as to make some people believe that it is contagious. Nearly every feed-lot in northern Colorado has experienced the malady. One of the most impressive outbreaks occurred near Johnstown, early in January, 1935. Seven steers out of 49 were affected at one time. They were on alfalfa, bean straw, beet-tops and pulp. The grain was composed of corn, barley and wheat ground together. In addition they were getting limestone and bonemeal. About ten days previous to the outbreak, the owner had felt that they were not eating enough of the mineral, so he made it more available and added some salt to make it more palatable. He said that they had recently been consuming increased quantities of it. One steer had been slaughtered and six more were showing evidence of pain, a dribbling of urine from the sheath and collection of salts on the long hairs around the prepuce.

The mineral was removed, the steers were put on corn and alfalfa, the sick ones were drenched with sodium bicarbonate and all made a nice recovery with no more cases appearing.

Nearly every winter, cases appear among the steers experimented on on the college farm. Rarely is there anything approaching an outbreak, but from one to three cases occur each year. This year, six cases developed within a period of 20 days. These animals were getting a basic ration of corn, cottoncake, wet beet pulp and alfalfa. The variable was beet-tops prepared in various ways and mixed with differing amounts of wheat straw. Those getting the tops were limited on their alfalfa. All cases have so far recovered when given sodium bicarbonate and having the ration withheld. It ought to be pointed out that the college feed-lots are supplied with exceptionally soft water, which constitutes the Fort Collins city supply, while most other feed-lots in northern Colorado are supplied with a water heavy in alkali salts.

Experimental Work

No experimental work done with cattle has come to our attention but there are several reports of work done with sheep. The earliest of these was from the Iowa station.[2] In 1910, that

station reported on work done to show the relation of sugar beets and mangels to the disease. Four lots were used, one getting hay and grain; one hay, grain and corn silage; one hay, grain and mangels; and one hay, grain and sugar beets. Three of the eleven sheep getting sugar beets died during the time of the experiment and showed calculi in the bladder. Two others showed calculi in the kidneys at the time of slaughter. While those on mangels showed no calculi, it was felt that the condition of the bladder and kidneys indicated a tendency to calculus formation. Since this work, the belief has become general that beets and mangels, and their by-products, bear a casual relationship to the formation of calculi in sheep and cattle.

The Iowa Station[3] also cast suspicion on molasses, reporting in 1923 that 3 per cent (1 lamb) of the check lot, 11 per cent of the cane molasses lot and 31 per cent of the beet molasses lot showed urinary calculi on slaughter.

This work was followed by that at the Indiana station[4] in 1931, where it was pointed out that root crops, because they increased the amount of urine, were less likely to result in calculi than the less watery feed, such as hay and grain. In one experiment at Purdue, the lambs on mangels, although they drank no water, passed on the average ten times as much urine as those on other feeds. This work incriminated cereal grains and particularly wheat bran. Quting from their report:

> When wheat bran is introduced into a ration it carries with it a large mineral content, especially phosphorus compounds. These readily form water-insoluble combinations with calcium, magnesium and aluminum, and gravel is deposited in the kidney and bladder, thereby often causing serious trouble to male sheep.

The Ohio Station[5] also found that sheep being fed heavily on phosphatic feed were more liable to the formation of calculi.

The Cause of Calculi

Water: Hard water has been more frequently offered to explain urinary calculi than any other factor. While it cannot be said that the theory has been disproved, it seems strange that no experimental results are given in support. Calculi do occur in animals on very soft water as well as on hard and apparently as frequently. More may be said on the amount of water consumed. Calculi are more common in winter when water consumption is limited. The experience in the feed-lots at Akron previously mentioned, where a veritable outbreak followed the freeze-up of the water supply, pointed in this direction. Theoretically at least less water results in a more concentrated urine and salts would more readily settle out in the bladder.

Vitamin A: Osborn and Mendel,[6] as early as 1917, reported a high incidence of calculi in their vitamin-A-deficient rats. McCarrison,[7] in an attempt to explain the prevalence of calculi in the native population of India, found that rats on a ration of whole wheat flour, linseed meal, corn flour, salt and calcium phosphate frequently developed calculi. Critics have pointed out that not only was this ration deficient in the A vitamin but it was also exceptionally high in phosphates. Japanese workers[8] and Higgins[9] at the Cleveland Clinic have confirmed the results of previous workers in producing calculi in rats on a deficient diet almost at will. McCollum[10] points out, however, that he has found rats with calculi after they had been getting as high as 5 per cent of butterfat and 10 per cent of cod-liver oil.

The Council on Pharmacy and Chemistry[11] of the American Medical Association recently surveyed the whole situation and concluded that the case for vitamin-A deficiency still lacked proof and they therefore refused to approve claims made for certain foods as preventive or curative agents because of their high vitamin-A content.

The fact that calculi in cattle and sheep at least are largely seen in the winter may argue in favor of the vitamin-A theory as well as the lowered water consumption.

The experience of the sheep-feeder who had such heavy losses after discontinuing the use of alfalfa may support also the vitamin theory, but here again several other factors entered, notably the increase in phosphates. Vitamin A is quite generally deficient in the rations in the drylands of eastern Colorado where calculi are so prevalent. However, most practitioners have seen the disease in both cattle and sheep on good alfalfa rations.

In an experiment in 1935, carried on by this station, vitamin A was so deficient that cottonseed poisoning developed in several of the steers, yet only one out of 80 showed calculi. The following year, on identical rations, two showed calculi although none showed evidence of cottonseed poisoning.

Mineral imbalance: McCollum felt that high calcium intake was responsible for much of the calculus formation in rats. Both the Indiana and the Ohio workers stressed phosphates as causative factors. Experience seems to indicate that both of these views may be correct. Possibly our wide use of lime in the rations of cattle during recent years may have increased the number of calculi. On the other hand, supplanting alfalfa with cane and making up the deficiency of protein with cottoncake, and especially feeding bran in place of corn, may not be without

their penalties. The wide use of mineral supplements during the past years, without knowing their composition or their need, may also have developed faulty metabolism in ways not yet understood.

Reaction: The deposition of urinary salts depends to a considerable degree on the reaction. Uric acid, oxalate and cystin calculi occur in acid urine and the phosphate and carbonate in alkaline. Since the urine of sheep and cattle is normally alkaline, phosphatic calculi are more common in these animals. Since the character of the diet determines the reaction, it is apparent that the food has its influence on the formation of calculi. Ketogenic and acid ash diets are believed by Higgins to have actually dissolved calculi in rats and in man. Some feeders feel that calculi may be dissolved in steers by withholding feed for a time. There might even be a scientific basis for this, since an animal living on its own tissues has an acid urine.

Hyperparathyroidism: Albright, Aub and Bauer[14] report that of 17 cases of hyperparathyroidism in man, ten showed the presence of urinary calculi. Their studies show that this disease of the parathyroids was associated with a high level of calcium and a low level of phosphorus in the blood and an increase of both in the urine. McCarrison also pointed out a connection between hyperparathyroidism and stone in the bladder. This relationship seems not to have been studied in the lower animals but seems worthy of some attention. Even though a relationship were discovered, there would still remain the effect of the other factors such as vitamin deficiency, mineral imbalance and diet on the parathyroids themselves.

Infection: Rosenow[12] and Hager and McGath,[13] at the Mayo Clinic, carried out work which strongly indicated that infection played a considerable part. The former inoculated dogs with streptococci which he had isolated from the teeth of animals affected with calculi and thus seemingly reproduced the disease. The latter, by direct injection of bacteria into the bladder of rabbits, succeeded in producing calculi. Infection seems to act by breaking down urea with the formation of ammonia, thus rendering the urine alkaline.

Fox men tell us that calculi are more prevalent during or immediately following the breeding season, which may indicate that infection is a factor in fur-bearing animals.

Infection is usually present in the bladder of all animals with calculi, but whether it is the cause or the result remains to be determined.

Urinary irritation: It was noted by Hager and McGath that bacteria did not implant themselves in the bladder unless the bladder was irritated. The cornification of the bladder epithelium that is commonly associated with vitamin-A deficiency may be explained on the basis of chronic irritation, and the association of a deficiency and infection is too well known to require further elucidation. It is quite possible that some foods carry a constituent that produces a low-grade urinary irritation and that, after a period of weeks or months, this sets up a sufficient degree of cystitis to cause the deposition of salts.

Composition

Most of the urinary calculi of horses are composed of calcium carbonate. Practically all analysts agree that the calculi of cattle and sheep are composed of calcium, magnesium and aluminum salts of phosphoric acid. The studies made by our own chemistry department confirm these views. Xanthine calculi[15] in sheep have been reported from New Zealand, but apparently from nowhere else.

Uric acid, oxalate and phosphatic calculi are reported in man, and the same varieties in dogs, although even in these latter animals phosphatic stones seem to predominate. A recent report from the veterinary school at Utrecht gives the composition of canine calculi in 39 cases as follows:

Phosphate	28
Phosphate and oxalate	8
Cystin	2
Uric acid	1

The indication here is that either vegetable or cereal diets are responsible for most of the calculi in dogs, or the reaction of the urine is changed as a result of infection.

A single analysis of a stone from a fox pup, made by our chemist, shows the composition to be wholly calcium and magnesium phosphate.

Cystin calculi have been described in cats.

Symptoms

The first symptom to be noted in either cattle or sheep is a restlessness and an occasional straining to urinate. In cattle there is a wiggling of the root of the tail and kicking at the abdomen. An examination at this time may reveal a dribbling of urine from the sheath. It will also almost invariably show a collection of precipitated salts attached to the wool or the hair surrounding the opening of the sheath. This is due to the evap-

oration of the urine that keeps this area wet. Later, the animal stands quietly, moves only with difficulty and then with a straddling gait. The appetite is suspended. If the bladder ruptures, the abdomen enlarges, due to accumulation of fluid. In this case death always follows.

Where the obstruction is in the urethra and a small amount of urine can seep through, necrosis of the area may occur, resulting in an extensive edematous swelling of the subcutis in this vicinity. The author observed one case where urine actually broke through the skin several inches back of the opening of the prepuce, the swelling subsided and recovery took place, the urine passing through this artificial opening throughout the rest of the feeding period.

Lesions

Following death from a ruptured bladder, the abdomen may contain a large quantity of blood-tinged fluid. The bladder will then be contracted into the pelvis and may even be overlooked. However, if examined, a rent will be found in it, surrounded by a hemorrhagic area. The mucous lining will show a hemorrhagic cystitis. The calculi themselves may be missed when the bladder is ruptured, since they never constitute anything more than a mass of sand-like grains.

An examination of the urethra will usually reveal a severe inflammation with swelling of the mucous membrane. The obstruction may be anywhere from the processus urethrae (in the sheep) to the sphincter of the bladder itself. The two most common sites are near the terminal opening and in the S-shaped curve. In some instances, however, the whole urethra is packed full and even operative procedures at the ischial arch fail because of this.

The kidneys are usually enlarged, the pelvis is dilated and the tubules, as well as the pelvis, may contain sand grains (calculi). The kidney may be mottled due to dilatation of certain tubules and to increase in fibrous tissue.

Treatment

Much attention has been given recently to Higgins' findings that in rats, having produced calculi by a vitamin-A-deficient diet, he could dissolve them by the addition of 2 drops of cod-liver oil daily. Recently, he has reported a successful outcome in 23 cases in man in which he used a high vitamin A and an acid ash diet. Oppenheimer and Pollack[16] failed to confirm these findings in 27 patients, stating that not a single calculus dis-

solved and that five of them actually increased in size during treatment.

Moussu seems to have been one of the first to recommend sodium bicarbonate for the treatment of phosphatic calculi in cattle and sheep. This treatment has been widely used in this country, sometimes along with sodium hyposulfite and in some instances with remarkable results. As long as any urine can seep through, removing the animals from feed and administering sodium bicarbonate may be effective. There are, however, many cases that will require surgery if the animal is to be saved.

Some report that removal of the urethral process in sheep may be sufficient. From our experience these cases are rare. Usually the urethra must be opened at the ischial arch and even then it may be impossible to pass a catheter into the bladder.

Prevention

While it cannot be said at this time just what factors are of most importance in causing calculi in cattle and sheep, experience seems to indicate that they are more prevalent during the winter and when the animals are on a dry roughage, deficient in vitamin A and high in phosphate. The addition of lime may also be a factor. Whether beet by-products are responsible is still controversial. Where sheep are eating large quantities of wet pulp they drink no water, but calculi are quite rare. The case against tops and molasses seems to be stronger. Bran, wheat and cottonseed cake are, to say the least, under considerable suspicion. Finally, a change of either cattle or sheep to corn and alfalfa has usually resulted in a cessation of the outbreaks.

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DIETARY DEFICIENCIES AND RELATED SYMPTOMATOLOGY IN DOMESTIC ANIMALS*

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INTRODUCTION

During the first generation of this century, extensive programs of research with markedly fruitful results have taken place within the broad field of physiology now recognized as the specialized field of nutrition. New concepts have been added to the subject, including vitamins, trace elements, and interrelationships of one essential with others.

The practitioner of veterinary medicine is far removed from the pure biological science laboratory. Nevertheless, time has abundantly revealed that great discoveries made in these laboratories with the drosophila fly, white rat, or guinea pig as the test animal will ultimately reach the ken of those called upon to advise regarding the well-being of our domestic animal population.

This is because new knowledge uncovered experimentally through these small species usually constitutes universal biological laws applicable to all animal life. The marked species variations complicate the picture seriously and require time for their elucidation; but the fundamental principles, necessarily worked out largely in these small laboratory animals because of their relatively short life cycles, still prevail as the scale of animal life is ascended. It brings into clear recognition the importance of properly coördinated functioning between the pure science laboratory, the applied science laboratory and those who are extending the application of knowledge to the field of economic live stock production and its relationship to human welfare including human health.

Knowledge is usually in advance of practice and it is fortunate that this is the case. Nevertheless, the public have become conscious of the value of research and are frequently clamoring at the doors of research laboratories for the latest news of the progress of certain types of investigations that bear on problems of wide economic importance.

There are often stages in the progress of elucidation on the frontier of knowledge when all of the links in the chain of neces-

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sary facts for complete understanding have not been obtained. Under these conditions that which appears to be truth may be only half truth. Such a condition may exist over a period of years in even a very narrow part of a problem where intensive work is being prosecuted. This is the period when shrewd commercialism has its inning and it proceeds to make hay while the sun shines, knowing full well the period for profit may be very temporary.

The field of nutrition during recent years has presented golden opportunities for this type of exploitation. It is logical, therefore, that a professional group such as represented by this Association should be confronted with more or less confusion. Some will join the exploitation program with no other thought than desire to make hay while the sun shines, or with a sincere belief that results prove the program sound. Others are reactionary or recognize the exploitation and will have no part in utilization of advancing knowledge. The majority are between the two extremes and in the general confusion wonder how to proceed.

As a group we should all readily understand the basic difference between foods and medicines. The former must be supplied to all animals throughout their lives and price limitations are serious. The latter are supplied to a relatively small percentage of animals for a short period of time and price may be no limitation or only a minor one.

It has been stated that the value of a food depends on the need for it in the diet. The prolonged absence of sufficient quantity of a dietary essential may have animals close to death and its value to these particular animals might be worth the full value of the animals because without it they would die. Under no circumstances could this be interpreted to mean that such a value and procedure would be economically sound for general application.

Place of Nutrition

With the above background let us proceed to recognize that nutrition is now conceded to play a very much more important rôle in the well-being of our animals than was recognized a generation ago. Then it was common practice among the best informed to balance a ration of roughage and concentrates on total digestible nutrients of proteins, carbohydrates and fats, fix the nutritive ratio depending on whether the animals were growing, fattening or producing milk, feed sufficient total energy for maintenance and production and consider the nutrition problem settled.

Today we recognize that nutrition is a very involved field of knowledge with new facts of wide importance being uncovered constantly and the end not in sight. The highly artificial conditions under which our animals are maintained in fenced enclosures and fed more and more the by-products of industry render their rations very far removed from that obtaining under natural conditions. Widespread recurring drouths of recent years add further complications and enhance the hazard of deficiency of intake of essential substances.

We must therefore not overlook the possible place of nutritional deficiencies as the etiological factors in abnormalities of live stock and give them a place along with bacteria, parasites and hereditary defects in the broad differentiation by a learned profession of abnormalities encountered in domestic animals.

It will be necessary, because of time limitations in going into more details, still to speak in general terms and limit the subject to minerals and vitamins. Under good management and normal feed conditions all essential substances are likely to be contained in the regular feed supply. Best husbandry practice is that which supplies a variety of foodstuffs in sufficient quantity to meet all requirements. The old idea of balancing a ration with feeds from five plant sources, while not the best from a research point of view, is to be commended more than ever for practical feeding in the light of present-day knowledge. Some understanding of the constituents of standard feeds is essential so that from examining the ration fed the possibility of essentials being lacking will be recognized.

It is rare for a single nutritional deficiency to be unaccompanied by the existence of others, particularly under more nearly natural conditions on the range. If progressive starvation is occurring, accompanied by several deficiencies, the manifestation of symptoms characteristic of the individual deficiencies may not occur because of the generally lowered metabolic rate. On the other hand, if such animals are supplemented with feed lacking in some essential so that normal energy intake is occurring for maintenance and growth or fattening, the deficiency symptoms will rapidly develop.

MINERALS

Calcium and phosphorus: These elements come in for the largest share of attention in this group of secondary dietary essentials, including their quantity relation to each other in the diet, the so-called calcium-phosphorus ratio. The latter has been a cause of controversy because of its recognized importance in

rachitis, in children and the susceptibility of the white rat to slight changes from the optimum. Evidence supports the desirability of having this ratio narrow. Meigs *et al*,[1] from their earlier experiments, suggested that an intake of two parts or more by weight of calcium to one of phosphorus in dairy cows constituted an excess which might interfere with phosphorus absorption. Further work by them and field observations since that time have now caused it to be quite generally recognized that ratios in cattle feeding as wide as 10 to 1 are not necessarily harmful. Ruminants are probably less sensitive to wide ratios than hogs. Nevertheless, extremes should be avoided over long periods of time.

Recognition should be given to the fact that those animals living largely on roughage—cattle and sheep—are liable to be short of phosphorus because stems and leaves of plants are low in this element and rich in calcium.

On the other hand, animals subsisting largely on concentrates —hogs and poultry—are liable to be short of calcium because seeds of plants and by-product feeds made largely from seeds, as bran, rice polish, cottonseed meal, linseed meal, etc., are rich in phosphorus and low in calcium.

The oxides of these elements are frequently used in expressing quantities in feeds and this is confusing. To convert CaO to Ca, multiply by 0.71, and to convert P_2O_5 to P, multiply by 0.44. In other words only 44 per cent of P_2O_5 is phosphorus while 71 per cent of CaO is calcium.

Bone meal, the most widely used mineral supplement, contains both calcium and phosphorus. When calcium is required, ground limestone or oyster-shell flour is recommended and widely used in poultry and hog feeding because they are the cheapest sources and the calcium content is as readily available as calcium from any other source. This latter point was in dispute some years back but has now become quite definitely settled. No such pure source of phosphorus is available for use where this element is lacking. Phosphorus is available as mono-, di-, and tri-calcium phosphate in which the percentages of calcium and phosphorus are as follows:

	Ca	*P*
Mono-calcium phosphate	14.10	19.30
Di-calcium phosphate	27.80	19.70
Tri-calcium phosphate (bone meal)	32.20	14.30

Bone meal is considerably the lowest priced and the increased phosphorus content of the other products does not justify their purchase for live stock feeding. They all contain considerable

calcium and are usually fed only where phosphorus is required. Thus, there is no reason to use other sources of calcium with them because it is contra-indicated. Despite this a very widely used mixture consists of 40 per cent bone meal, 40 per cent limestone and 20 per cent salt. The sodium phosphates are available and constitute a satisfactory source of phosphorus, but the price is relatively too high for general use.

In range cattle on natural vegetation, when the feed is green and young, no mineral supplements are needed. As the season advances and the plants mature and dry with seed shattering, phosphorus which goes out with the seeds becomes more and more reduced. Therefore, while one part of bone meal to three of salt will be satisfactory in the early summer, three parts of bone meal to one of salt will be preferably late in the grazing season. Feed on the range, particularly where annual plants are the main source of forage, develops multiple deficiencies as the season advances, of which, in addition to phosphorus, protein, vitamin A and low total energy intake are most important.

Low phosphorus intake is first manifested by reduced appetite and low phosphorus content of the blood-serum. The degree to which additional phosphorus will increase the ingestion of the poor range feed is the extent to which it can aid the low protein intake and this is very limited. When protein supplement is supplied in the form of cottonseed cake, for example, this contains both protein and phosphorus and, given at the rate of 2 or 3 pounds per head per day, renders the phosphorus alone no longer needed. Maynard *et al*,[2] at the Utah Station, showed that steers with low blood phosphorus from Rocky Mountain ranges, when placed in the feed-lot on a balanced ration of alfalfa hay, beet pulp and beet molasses, made better gains when cottonseed meal was added. Later, it was found that this advantage was due to the high phosphorus content of the meal and equally good or better results were obtained with much smaller quantities of bone meal at a net saving in feed cost per animal of $9.00 to $13.00.

Feeding mineral supplement to all farm animals is unnecessary and even high-producing dairy cows on legume hay and concentrates get along well without them.

On the other hand, unusual feeding practices necessitated by economic conditions or vagaries of the weather, such as drouth, may result in serious deficiencies.

Shortage of calcium in cattle is relatively rare but under prolonged feeding of grain hay and certain concentrates the low intake of this element may result in symptoms. Tenderness in the

front feet so readily manifested in hogs may occur to the point that animals will stand resting on their knees.

Milk fever caused by low blood calcium is not a true calcium deficiency. It results from the temporary failure of the high-producing cow to marshal the calcium from the body stores in the skeleton to the blood-stream as rapidly as it is removed by the udder. When the blood calcium drops to meet the demands of milk secretion, the cow goes into coma. McCampbell,[3] at the Kansas Station, found that fattening cattle on a balanced ration of corn, cottonseed meal, sorghum silage and prairie hay made faster and more economical gains if 2 pounds of alfalfa hay replaced the prairie hay. The value of the alfalfa hay was shown to be due to its high calcium content, and by feeding 1/10 pound of ground limestone with the prairie hay equally good results were obtained at less cost.

Excess of these elements, within reason, is harmless as they pass out of the intestinal tract unabsorbed. The exception to the general rule at present is one form of perosis or slipped tendon in poultry. This is involved with excess of phosphorus but is also complicated with manganese intake. Jukes, at the California Station, has classified four etiological factors in this widely discussed condition in poultry pathology: (1) strict confinement, as in battery brooders; (2) heredity; (3) a phosphorus content above 0.7 to 0.9 per cent in the ration; and (4), the protective factor against its appearance in rice bran, wheat bran and oat hulls, recently found by Wilgus, at Cornell, to be the element manganese.

Iron and associated elements: Deficiencies resulting in anemia in farm animals are probably more widespread than generally believed. The form in young pigs kept on board or cement floors during nursing is well known and supplying the necessary iron by painting the udders of the nursing sows with iron preparations is widely practiced with good results.

In anemia, iron, copper, cobalt and manganese may all come in as possible etiological factors. The latter three are among the trace elements required in nutrition. Bush sickness in sheep in New Zealand and other parts of the world is a form of anemia thought to be due to lack of iron. The successful results of iron treatment were found to be due to relatively high contamination of the iron medication with cobalt. Pasture plants readily take up sufficient cobalt if it is present in the soil. Livers of healthy sheep were found to contain 0.15 to 0.25 p.p.m. compared to 0.02 p.p.m. in those of affected sheep. Very minute quantities of co-

balt were effective in preventing and curing the condition as reported by Denham.[4] There is incomplete evidence to show that dirt-eating by farm animals, which is widespread in some areas, is associated with these deficiencies. This may account for the unexplained practice of feeders of long experience supplying fresh dirt in the feed bunks to cattle fattening in dry lot. The existence of the condition has been definitely established in cattle on the white and gray sandy soils of Florida. Extensive investigations on the problem have been carried on by workers at the Florida Experiment Station.

Iodine: Shortage of this element is very important in certain sections of this country now pretty well delimited. The symptoms in affected animals, consisting of the birth of dead or weak, hairless or goitrous offspring, are so characteristic that diagnosis is comparatively easy. Symptoms of iodine deficiency in man are manifested over much wider areas than is the case with live stock. This has been observed in this country and also in Switzerland, which has so far baffled explanation. Conditions surrounding the closely delimited areas where the trouble exists are sometimes so peculiar that question arises as to whether or not other factors limiting availability are involved in its manifestation rather than iodine deficiency alone. In Shasta County, California, for example, there is well marked evidence of the trouble in cattle, hogs, sheep, dogs and humans along Hat Creek, which has its origin on the south side of the base of Mount Lassen. Rising River comes out of the lava beds as an enormous spring on the eastern side of the Hat Creek drainage area and maintains a very even volume of water throughout the year. It flows through Hat Creek Valley for a distance of about four miles, gradually converging with and finally flowing into Hat Creek. There has been no evidence of iodine deficiency along Rising River or along Hat Creek below the point where Rising River becomes confluent with it. Iodine determinations of the waters of these two streams have not been made, but the abruptness with which the affected area terminates is interesting. This same condition exists in other iodine-deficient sections.

In supplying iodine for this condition the limitation of iodized salt blocks should be considered. Under field conditions the iodine volatilizes out of the salt about ½ inch below the surface licked by the animal and this continues as the block is consumed, so that animals do not get the required amount of iodine and losses will continue.

Potassium iodide, added to ground or half-ground salt in the proportion of 1 ounce to each 100 pounds, is preferable to salt

bricks and where acidity is liable to develop and lead to rapid freeing of iodine from potassium iodide the use of potassium iodate is recommended.

VITAMINS

The vitamin deficiency group of abnormalities has now been extensively studied in domestic animal species under both experimental and natural conditions.

Vitamin A: This has assumed very great economic importance. Lack of this essential is to be suspected when symptoms develop after more or less prolonged feeding on rations lacking green or yellow color; concentrates, for example, except yellow corn, roots except carrots and grain hay or any roughage including alfalfa which has been bleached until all green color has been lost.

The source of this substance is the pigment carotene present in all green plants but masked by the green chlorophyll pigment. It is changed more completely into vitamin A, which is colorless, in some species of animals than others. It is stored in the animal in the liver and body fat and is the yellow color in milk of Guernsey and Jersey cattle. Holstein milk contains just as much of the protective substance when the cows are on the same feed as Guernsey or Jersey milk, but it is in the form of vitamin A, which is colorless. Color of fat in beef animals is involved with this factor and the reason grass-fattened cattle have yellow fat as compared to the relatively white fat of dry-lot-fattened cattle. The color in beef fat is discriminated against by price reduction, while in milk premiums are paid for good rich color. Sheep and hogs convert all ingested carotene into vitamin A and their fat is white.

Guernsey and Jersey body fat and milk fat can be made as white as lard by withholding carotene from the diets of these animals and supplying the necessary preformed vitamin A in the form of cod-liver oil.

Color of egg yolk is associated with this pigment and also xanthophyll, another yellow pigment of green plants. It is somewhat of a problem to produce eggs with pale yolks as desired on the New York egg-market and still keep heavily laying hens free from symptoms of vitamin-A deficiency, which is the reason cod-liver oil is fed to poultry producing premium quality eggs. The above may seem to be very mild alterations but are factors of real interest and great economic importance.

Other evidences which may more definitely be classed as symptoms from subminimum intake in domestic animals are very varied and may involve epithelial cell structures in any part of

the body. Storage of the vitamin varies with age and species. Growth, reproduction and lactation are greater drains on storage than maintenance after development has ceased. This accounts for the time variations in the development of symptoms in different animals when on subminimum intake.

Night blindness is probably the first symptom of the deficiency in all species but is overlooked unless special tests are made to demonstrate its presence. Its existence can be demonstrated in twilight or after darkness by a dim light, from a stable-lantern or electric globe, in which the suspected animals are forced to move around objects such as feed-troughs or supporting columns. Affected animals will run head on into an obstruction and into each other, while normal animals moving with equal speed will readily avoid any objects in their paths.

Another eye lesion is xerophthalmia, so widely described in the laboratory white rat, where it is practically a constant symptom. This is caused by keratinization of the cornea due to drying from involvement of the tear-glands and secondary invasion with bacteria. It is not so regularly present in farm animals, probably because excessive lacrymation is common but does occur under both experimental and natural conditions and in cattle requires some care to differentiate it from infectious keratitis. Ulceration of the cornea in dogs may be due to this cause. These lesions are temporary and readily heal on administration of the vitamin.

Another eye manifestation of the condition, particularly in cattle, is complete and permanent blindness without visible change in the eye-ball, a form of amaurosis which results from degeneration and shrinkage of the optic nerves where they pass through the optic canals in the sphenoid bone. There is also evidence of constriction of the bony structure and this has caused some discussion as to which is the primary condition. We are of the opinion that the nerve change is primary, because in experiments with cattle we have produced marked degenerative changes resulting in shrinkage of the optic nerves throughout their length.[5] Animals may be born blind from this condition and new-born pigs were experimentally produced at the Texas Station with no eyeballs, by having the sow depleted at the proper time in gestation.

In all animals depletion of body stores in pregnant females results in death of fetuses *in utero* or the birth of weak offspring which frequently die soon after birth. This may be the only symptom of the condition manifested in range cattle.

The air-passages may become involved, resulting in lung abscess or a chronic form of pneumonia. Diarrhea is not uncommon and in new-born calves may be confused with white scours.

Degenerative changes in the kidney may occur and also in the genital tract. Ability of males to breed and females to come in heat continues until very advanced stages of the deficiency, and alterations in the genital tract are not permanent as they are in the male deprived of vitamin E over too long a period.

Convulsions in cattle and hogs occur and death may result suddenly in these seizures. Paralysis in hogs is probably of varied etiology, but one form of the condition results from deficiency of this vitamin. Prior to the development of paralysis, there is unsteady gait from incoördination of movements and the back muscles of one side may become involved, producing marked lateral curvature of the spine.

Vitamin B: There are a number of specific factors included under this vitamin in addition to the thermostabile factor now classified as vitamin G. Some species of animals, ruminants for example, can synthesize this substance by bacterial action in the gastro-intestinal tract. Symptoms of its deficiency in mammals are not very well established although evidence is accumulating that it bears an important relation to certain abnormalities and retarded growth curve, particularly in swine under nutritional régimes lacking dairy products, green feed, or other plentiful source of the substance. Values of dairy by-products, particularly butter milk, skim milk, and whey, may partly be ascribed to their high content of water-soluble vitamins.[6] Where they are included in rations for swine, necessary food intake per pound gain in weight is reduced below that required for most other rations.

While the polyneuritis of fowls fed on polished rice was the original condition that caused the discovery of this vitamin and is a very definite syndrome, it rarely occurs under natural conditions the way poultry are fed in this country.

Eating of feces, technically termed copraphagia, is a symptom generally ascribed to the B-complex deficiency, since the refection experiments in rats were reported by Fridericia *et al.*[7] The feces contain this substance through bacterial synthesis in the lower bowel. Range cattle on dry feed, turned into a small holding pasture where horses have grazed, will sometimes proceed to consume horse manure with avidity, which form of depraved appetite is not readily explainable if all of the water-soluble factors are synthesized in the rumen and copraphagia is always a symptom of their deficiency.

Vitamin D: This vitamin is associated with bone and tooth development and the syndromes rachitis and osteomalacia. It is

therefore closely correlated with calcium and phosphorus metabolism and the ratio of these elements in the diet. Hay, during exposure to sunlight in the curing process, has its vitamin D increased by the activation of sterols contained in it by the ultraviolet sun rays, while its vitamin-A content is being reduced through the oxidation of its contained carotene pigment.

Bone changes are quite frequently seen in dogs and poultry and are occasionally seen in calves raised under limited sunshine from too close housing.

Osteomalacia or creeps in adult cattle is not uncommon in dairy herds in some sections of the country. Its etiology is still in a very clouded state. It practically always develops in heavy milking cows but only one or two cases will develop at a time in a herd of heavy milkers, all kept under the same conditions. This may continue over periods of years with or without the addition of mineral supplements and in areas with abundant sunshine through a very large part of the year. Valuable affected animals that fail to get with calf and have a very long dry period, particularly on green feed, will make complete recoveries, while in others no improvement will be observed. Sometimes moving animals relatively short distances to new pastures will have a curative effect.

The public-health side of vitamin D is becoming very important because of the various methods now commercially practiced, to increase the potency of milk in this factor.

Thus, we have metabolized vitamin-D milk from cows which have been fed irradiated yeast, and irradiated vitamin-D milk, the result of treating the milk in a thin film to irradiation from an ultraviolet lamp; also the addition of vitamin-D concentrate directly to the milk and the now-abandoned procedure of irradiating the cows directly.

Some work has been done in monkeys to show that possible harmful effects may result to the arterial walls from overdosing with vitamin D.[8] Milk is the best source of calcium in the human diet and vitamin D is associated with efficiency of calcium utilization. These methods of producing special purpose milk are costly and in many cases are accepted for the commercial advantage they may give over competitors. The mixing of commercialism and human health problems needs to be viewed with care and there are those who believe that too artificial means of increasing the biological value of such a universally used and so valuable a food as milk may ultimately result in more harm than good. Nevertheless, opposition to these particular forms of in-

creasing the vitamin-D content of milk seems to be decreasing and in the neighborhood of 3 per cent of market milk is said to be so treated at the present time.

An unclassified, fat-soluble, antihemorrhagic vitamin relieving tendency to hemorrhages in chickens due to delayed blood-clotting has been reported independently by workers in this country (California Station) and Europe. This factor is stable to heat and light and transferred to the chick by way of the egg-yolk. It is present in hog-liver fat, hempseed, tomatoes and kale and is synthesized in fish meal allowed to putrify slightly. It can be extracted from alfalfa meal to the extent that two parts per million in the feed will protect against hemorrhages in a diet that would otherwise produce the condition.

Vitamin E: This is the antisterility vitamin thoroughly studied in rats. Manifestations under natural conditions have not been demonstrated in domestic animals. Wheatgerm oil, a rich source of the substance, is being recommended as a procedure to improve low fertility in stallions.

Vitamin G: This vitamin is taking on greater importance as knowledge regarding it develops. The nomenclature at present is very confused. It was originally included with vitamin B. When it could be definitely separated from the true B heat-labile polyneuritis factor by the application of heat, it was termed B_2 or the thermostable factor. Later, it was given a place in the alphabetical list as vitamin G. Chemical investigations showed the substance to be flavin and this could be obtained in concentrated form from milk (termed lactoflavin) and from liver (termed hepatoflavin). Investigations with pellagra in man and its analogue, black tongue in dogs, showed it was a deficiency allied with the G complex but this was not agreed to by all investigators and the term P.P. (pellagra preventive) factor was applied to it.

More recently, the term filtrate factor or the chick anti-dermatitis vitamin, also strongly growth-promoting, has been used in describing a water-soluble substance closely associated with vitamin G. This substance passed through filters of Fuller's earth on which the flavin is adsorbed. Both the filtrate factor and flavin are present in pork liver. It would therefore seem that vitamin G consists of more than one factor like the B_1 from which it was originally separated. Certainly at least two factors in the G complex are required by poultry and both of them are strongly growth-promoting. Cane molasses is a cheap concen-

trated source of the filtrate factor. There has been some work done to show that there are two antidermatitis factors and therefore this term should not be considered synonymous with the filtrate factor, especially since lactoflavin will cure dermatitis in turkeys and will neither prevent nor cure it in chickens.

In conclusion, attention is called to the ability of animals to store certain essential vitamins and minerals to a much greater extent than others. Vitamin A may be stored in sufficient quantity to be utilized over a period of six months or longer in some species on subminimum intake with no symptoms. On the other hand, symptoms in certain species will be manifested within two weeks, when one or more of the water-soluble vitamins are below requirements because storage facilities are very limited. Cattle can subsist on subminimum phosphorus intake for upwards of a year when stores are high with no manifestation, while little pigs will die from anemia before the eight-week nursing period is completed if their mother's milk is the only source of iron.

This brings into importance the supplying of a reasonably complete diet at all times. The practical value of this can readily be demonstrated by supplementing poor range feed early with a little supplement before the animals have become too depleted. Such animals will winter well compared to others which are left to border on starvation before supplements are supplied. Profitable live stock management does not include allowing animals to border on starvation or failure to supply sufficient quantities of essential substances until symptoms appear.

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Discussion

Dr. H. E. Moskey: I would like to ask Dr. Hart's opinion of the propaganda of the Iodine Educational Bureau, that all live stock feed needs supplementing with iodine for maximum milk production and maximum growth, and maximum egg production and maxmium growth in poultry.

DR. HART: Of course, this is a part of the propaganda which, even though done in high circles, causes a lot of trouble for the reason that it makes many think the whole thing is propaganda. The Iodine Educational Bureau has gone to extremes in this and other countries of the world. On the other hand, in those areas where iodine deficiency exists, it is a wonderful thing to see the complete cures that result. We are not going to get anywhere by feeding all animals iodine, just as we are not going to get anywhere by feeding all animals a mineral mixture. There is no use of recommending any $150- or $200-a-ton proprietary material, because the price is excessive. It is food essentials that we are supplying and live stock owners cannot afford to pay the price for highly advertised mixtures, the ingredients of which can be obtained for very much less money.

DR. SILAS KEMPF: In recent years especially, I have had quite a number of cows at the time of parturition where the hind quarters would just "double-cross." They were unable to support weight unless we would steady the animal, and then sometimes they would not support themselves either. Is that a vitamin-A deficiency, or is that something else?

DR. HART: I suppose you refer to the so-called preparturient paralysis, which is different in cattle than in sheep. There is a condition in cows carrying a calf, and we think it is involved in her nutrition somewhat, in which there is a relaxation of the ligaments so that the backbone presses on the spinal cord. Such an animal may go down and be completely paralyzed in the hind quarters, or she may be just wabbly. If such an animal is properly cared for until after she calves, there will be an increase in the tone of those ligaments with the weight of the calf removed, and she will make a complete recovery. There is nothing wrong with that animal except a mechanical defect of the bone structure. It is thought to result from relaxation of the ligaments, but we also know that animals may be short of certain minerals and this may cause the junctions of the bones with the ligaments to become softened and the ligaments thus tend to pull away.

This is demonstrated in the so-called "tennis arm" occurring in tennis players from pulling the ligaments at the junction where muscles attach to the bone. It is caused by the rotating motion in the tennis game, and we have similar conditions in animals, of which this thing may be an example. We have posted a couple of cows with this condition and did not demonstrate a lesion in the spinal cord. There does not seem to be a lesion there, even though the cow is paralyzed, but if these animals are taken care of, many of them will get up and walk.

DR. C. H. CASE: Do you think that vitamin-D milk produced by feeding yeast is beneficial for all children?

DR. HART: Yes, I think it is. The trouble is with these people who want to go to extremes. They strip their babies and have them tanned from head to foot with ultraviolet light or sunshine. In addition, they feed them haliver oil and irradiated or metabolized milk, and thus overdo the vitamin-D question in those children. When we ingest vitamin A in large quantities, we probably do no harm, because it is readily oxidized. Irradiated sterols are much more permanent, and maybe youngsters can stand it, but excess should be avoided and especially is this so in adults.

I do not believe we should stop irradiated milk, but I believe that in making a medicine out of milk we may do more harm than good, and I do not know how long a time will be required before we know whether we are doing good or bad. In children, not getting enough vitamin D, all well and good, but in California we think the children are getting enough Vitamin D, so they do not have to have so-called irradiated or metabolized milk. I will admit that opposition to it is reducing and it

is being widely used. Three per cent of the market milk in this country is being irradiated or metabolized.

DR. CASE: It takes three quarts of one to equal one of the other.

DR. HART: That is true, and if the youngster is short of D, it is one way to supply the deficiency, but he may not be short of D.

DR. K. K. SHOTT: Do you think that dirt-eating is an iron deficiency?

DR. HART: I would not want to be misunderstood on this dirt-eating proposition. It seems that if we give them something that may contain iron, cobalt, manganese or copper, they will tend to stop doing it. We have not sufficient evidence to know. This dirt-eating has been an interesting thing in live stock feeding for years and many practical dry-lot feeders supply it regularly. It was the laughing stock of trained men for a long time. We have ceased laughing about it now and are trying to find out something about it. We consider that when animals eat dirt, they are short of something that dirt may contain. They always eat dirt from certain places. We drive cattle for miles across the range, and when they get to a certain spot on the range, they lick dirt.

It has been only recently that cobalt and manganese have become involved. This dirt-eating is not far enough along to make a statement for all of you to go on. It is probably a deficiency, and at this time we suggest it is most likely one of the four elements that I mentioned.

DR. SHOTT: In our section of the country, we have quite a lot of lambs which die through this dirt-eating.

United States Civil Service Examination

The United States Civil Service Commission announces an open competitive examination for the position of Scientific Aid (Parasitology). Applications for this position must be on file with the Commission at Washington, D. C., not later than April 25, 1938 (east of Rocky Mountains), or April 28, 1938, for western points. Full information and application blanks may be obtained from the United States Civil Service Commission, Washington, D. C., from the secretary of the Board of U. S. Civil Service Examiners at any first-class post office, or from any United States Civil Service district office.

Man o'War Celebrates Birthday

Man o'War, America's greatest horse, celebrated his 21st birthday on March 29 by welcoming a daughter, foaled by Brushup, at the Faraway Farm of Samuel D. Riddle at Lexington, Ky. The new filly is a full sister of War Admiral, greatest son of Man o'War and the unbeaten three-year-old champion of 1937. Another famous son of "Big Red" is Battleship, winner of the Grand National Steeplechase at Aintree, England, recently.

COLIBACILLOSIS IN YOUNG CHICKS*

By CHARLES R. DAVIS, *College Park, Md.*
Live Stock Sanitary Service, University of Maryland

Organisms of the *Bacillus coli* group occasionally are found to be pathogenic and the cause of much trouble, particularly in young stock. Calves, pigs, foals, lambs and other young animals may show *B. coli* infection as an acute or chronic gastro-enteritis or as a septicemia. Apparently the intestinal mucous membranes are most permeable in newborn animals and especially so if they have suffered from poor nutrition or debilitating conditions. When highly pathogenic types become established on the premises, losses from retarded growth, if not death, result until corrective measures are instituted. Poultry may also become infected with pathogenic organisms of the *B. coli* group and, as in the larger animals, the young are most frequently affected.

During the early spring, chicks were received at the Maryland Live Stock Sanitary Service Laboratory from four different flocks at various times and records indicated that all of the chicks were purchased from the same hatchery. The history and symptoms were quite typical of pullorum disease, as the chicks started to die soon after being received and losses continued for ten to 14 days. By the time they were ten days old, from 15 to 40 per cent had died, many of the chicks were droopy, ate little if any food and showed a pasty vent. Inasmuch as the hatchery was a reliable firm and the source of eggs was from birds officially blood-tested for pullorum disease, it was at first considered to be a "break."

However, further examination of the 19 chicks submitted from the four widely scattered flocks, but all coming from the same hatchery, excluded pullorum from consideration. All chicks were alive on arrival at the laboratory and were killed just prior to making autopsies, cultures and microscopic examinations. The livers were about 50 per cent larger than normal, were mottled and contained numerous small white spots. Some few were covered with a tightly fitting film of a whitish exudate, gelatinous in consistency. The hearts seemed slightly enlarged and the pericardial sac in five instances was markedly distended with an amber-colored fluid. At the time of autopsy, the chicks were seven to ten days of age, yet little, if any, of the yolk sac had been absorbed. Cultures made from the liver, heart-blood and

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unabsorbed egg-yolk produced a heavy, uncontaminated growth of *B. coli communis.*

Suspicion, of course, was cast on the hatchery as being the source of infection. When inquiry was made, the owner reported that he had received numerous complaints of losses with symptoms similar to those seen in the birds examined in the laboratory. He had also noted his chicks were subnormal in size, although hatching eggs of standard weight were used. The percentage of hatchability was below normal and many chicks failed to emerge after pipping the shell. An unusually large air-cell was shown when the eggs were candled after seven and 14 days of incubation. The incubators were old, poorly ventilated and operated in a windowless barn. The machines so completely occupied the space that there was insufficient room available for proper handling and cleaning.

It was quite evident that dehydration was taking place because of the large size of the air-cell; the high percentage of chicks unable to escape from the shell and the small size and weak appearance of the birds hatched. Their weakened condition made them readily susceptible to the pathogenic colon organisms that had become established in the hatchery. Some improvement was noted in the hatchability and quality of the chicks when better ventilation and humidity were established. Losses, however, continued at such a high rate that new machines and better housing were imperative. When these new incubators were properly operated, normal, vigorous chicks were hatched and losses from *B. coli* infection ceased.

Prior to the purchase of the new machines, six chicks that had pipped the shells but had been unable to emerge and eleven chicks in unpipped eggs were secured, killed and cultured. *B. coli communis* was isolated from the liver of one chick that had pipped its shell but cultures from the remaining 16 produced no growth of any organisms. For experimental purposes 44 chicks were obtained from the hatchery having *B. coli* infection, which for convenience will hereafter be referred to as hatchery 1, and 19 were purchases as controls from another hatchery hereafter listed as No. 2. Assuming the eggs set in both hatcheries to be of average size, the differences in weight of the chicks hatched is significant and is shown in table I.

In dividing the chicks into groups for experimental purposes, as nearly as possible, the same number of each breed was used in each group.

Group 1: Three chicks from hatchery 1 and two from hatchery 2 were injected subcutaneously with 0.25 cc of the original

TABLE I—*Differences in weight of the chicks from two hatcheries.*

BREED	WEIGHT (GMS)		DIFFERENCE	
	HATCHERY 1	HATCHERY 2	WT. (GMS)	PER CENT
Single-Comb White Leghorn	33.8	39.0	5.2	13.4
Barred Plymouth Rock	38.7	44.8	6.1	13.6
New Hampshire	38.5			

B. coli culture. The density of the suspension was 50 times that of McFarland's nephelometer tube 1. The following morning, all three chicks from hatchery 1 were dead. There were no apparent lesions, but pure cultures of *B. coli communis* were isolated from the liver and heart-blood of each chick. The two chicks from hatchery 2 died on the ninth and twelfth days, respectively, after injection. The chick dying on the ninth day showed no lesions, but the chick that died on the twelfth day had peritonitis, pericarditis, liver lesions and unabsorbed egg-yolk. Cultures from both chicks were positive for *B. coli communis*.

Group 2: Six chicks from hatchery 1 and three from hatchery 2 were fed a heavy emulsion of liver and heart-blood from which *B. coli communis* had been isolated. Three chicks from hatchery 1 died on the fourth, sixth and ninth days, respectively, all of which had typical liver lesions and unabsorbed egg-yolk. *B. coli* was isolated from all three. The remaining three and the three from hatchery 2 showed no evidence of illness.

Group 3: Twenty-one chicks from hatchery 1 and eight from hatchery 2 were fed heavy suspensions of the *B. coli* culture on three consecutive days. Ten of the 21 from hatchery 1 died at the following rate: one on the first day, one on the third, two on the fourth, two on the fifth, two on the seventh and two on the eighth day. All had the typical liver lesions found in this condition as well as the unabsorbed egg-yolk. *B. coli* was recovered from the internal organs of all ten chicks. None of the eight chicks from hatchery 2 were affected.

Group 4: Twelve chicks from hatchery 1 and six from hatchery 2 were saved as controls. Four of those from hatchery 1 died, two on the fourth day, one on the 15th and one on the 16th day. All except the one that died on the 15th day had hepatitis, pericarditis and unabsorbed egg-yolk characteristic of this infection and *B. coli* was recovered on all cultures. The chick that died on the 15th day showed only an unabsorbed egg-yolk and

cultures were negative. None of the six chicks from hatchery 2 were affected. Table II shows the results in the four groups.

On the 42nd and 79th days, blood samples were drawn from three of the chicks remaining in groups 2, 3 and 4. Agglutina-

TABLE II—*Results of experimental inoculation of chicks.*

GROUP	HATCHERY	CHICKS	DIED	DAYS BEFORE DEATH	B. COLI	LESIONS
1	1	3	3	1	Pos.	None
	2	2	2	9–12	Pos.	Ninth day, none. Twelfth day typical*
2	1	6	3	4–6–9	Pos.	Typical in all
	2	3	0			
3	1	21	10	1–3–4–4–5–5–7–7–8–8	Pos.	Typical in all
	2	8	0			
4	1	12	4	4–4–15–16	3 Pos. 1 Neg.	Typical in all
	2	6	0			

*Typical lesions were pericarditis, unabsorbed egg-yolk, enlarged liver containing numerous small white spots.

tion tests were run using both *B. coli* and *Salmonella pullorum* as antigens. These results are shown in table III.

SUMMARY

Pure cultures of *B. coli communis* were obtained on autopsy of 19 chicks originating from the same hatchery but coming from four widely scattered flocks. History of all four flocks and lesions found in all 19 chicks strongly suggested pullorum infection, but cultures were negative for this disease. Inspection of the hatchery indicated improper incubation, and lack of ventilation caused poor hatches of small, weak chicks. Subcutaneous injection of *B. coli communis* in these weak chicks resulted in death in less than 24 hours. Similar injections in chicks from another hatchery required several days to cause death. Six chicks from hatchery 1 and three from hatchery 2 were fed an emulsion of liver and heart-blood from a *B. coli*-infected chick. This resulted in a 50 per cent mortality in the six from hatchery 1 while those from hatchery 2 were not affected.

Twenty-one chicks from hatchery 1 and eight from hatchery 2 were fed a heavy suspension of the *B. coli* culture. This resulted in 47.6 per cent mortality, with typical lesions of this

TABLE III—*Results of agglutination tests on chicks.*

	Age	42 Days				79 Days			
	Antigens	B. Coli		S. Pullorum		B. Coli		S. Pullorum	
	Dilutions	1:25	1:50	1:25	1:50	1:25	1:50	1:25	1:50
Group 2 (Fed emulsion)	Hatchery 1	+	+	—	—	—	—	—	—
		P	—	—	—	—	—	—	—
		P	—	—	—	—	—	—	—
	Hatchery 2	—	—	—	—	—	—	—	—
		—	—	—	—	—	—	—	—
		—	—	—	—	—	—	—	—
Group 3 (Fed *B. coli* cultures)	Hatchery 1	+	+	—	—	—	—	—	—
		+	+	—	—	—	—	—	—
		+	+	—	—	—	—	—	—
	Hatchery 2	—	—	—	—	—	—	—	—
		—	—	—	—	—	—	—	—
		—	—	—	—	—	—	—	—
Group 4 (Controls)	Hatchery 1	+	+	—	—	—	—	—	—
		—	—	—	—	—	—	—	—
		+	P	—	—	—	—	—	—
	Hatchery 2	—	—	—	—	—	—	—	—
		—	—	—	—	—	—	—	—
		—	—	—	—	—	—	—	—

infection in the chicks from hatchery 1. The eight from hatchery 2 were not affected. Twelve chicks from hatchery 1 and six from hatchery 2 were kept as controls. There was a 25 per cent mortality in the chicks from hatchery 1, with typical lesion and positive *B. coli* cultures from three of the four chicks. Six chicks with pipped shells and eleven with non-pipped shells from hatchery 1 were cultured. One chick with a pipped shell gave a pure culture of *B. coli communis* but cultures from the remaining 16 were negative.

CONCLUSIONS

1. Eggs hatched in a faulty incubator having insufficient moisture and improper ventilation resulted in a poor hatch of chicks with low vitality.

2. *B. coli communis* may become pathogenic for chicks having a low vitality.

3. A correct diagnosis cannot be made on the history of a flock and the lesions of chicks. Cultures must be made and identified to eliminate confusion with pullorum infection.

THE VETERINARIAN'S DUTY IN STANDARDIZING DAIRY INSPECTION*

By WALTER HYDE MARTIN, *El Reno, Okla.*

The real place to start in the standardization of milk sanitation, on a nation-wide basis, is in the training of the individual who does the actual inspection.

Before entering into the subject matter of this paper, permit me to present a brief outline of the veterinary organizations of this country. In most ways, we have a well-organized service in all states. Undoubtedly the United States Bureau of Animal Industry has the best organized veterinary service in the world. We think this has been demonstrated by its accomplishments, when compared with those of other nations. Next in line comes the state control services, followed by the American Veterinary Medical Association, the United States Live Stock Sanitary Association, and the state organizations. Through these various branches of veterinary service, the control of animal diseases is well in hand, as demonstrated by the control of bovine tuberculosis and the eradication of foot-and-mouth disease, pleuro-pneumonia and glanders. The Bureau service has an organized field force as well as a perfectly organized system of meat inspection directly controlling every department of the meat-packing industry.

To the veterinary profession must go the honor for the control of tuberculosis in cattle and to what extent it may be transmitted through milk and meat to man. Through these effective control measures millions of dollars have been added annually to one of the nation's greatest industries, but a far more important result has been the benefit to the health and well-being of the people generally. No other nation has considered this control measure possible until within the last few months, when Great Britain made a substantial appropriation for the control of bovine tuberculosis in Scotland and Wales.

Veterinarians have been given highly deserved credit for their efficient work in the control of diseases transmissible to animals as far as these affect other animals. Now we have demonstrated that we have been of service to society as a whole in protecting man from becoming infected by these transmissible diseases of animals. When the health service of this nation, in which the

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medical profession plays the most important part, recognizes the close relationship between human and veterinary medicine, then the veterinarian will receive the recognition due him. In other words, this line of demarcation between human and veterinary medicine must be erased. Permit me to suggest that a veterinarian should be on every state board of health for giving counsel, if for no other reason, because problems present themselves daily which the modern veterinarian alone can answer.

Dairy Inspection

Returning to the veterinarian's duty in standardizing dairy inspection: Veterinarians, in conjunction with the proper sanitary service, should challenge the manner in which dairy inspection is now carried on in its haphazard, happy-go-lucky sort of way in municipalities that are not under government control. It is partly in the hands of untrained and unskilled inspectors, a large percentage of whom come from the laity without any comprehension of what real dairy inspection means to the public health of a community. Under this method of inspection we have seen milk-borne epidemics which have cost the lives of numbers of people in municipalities through the country.

The public as well as the inspectors need to be educated. Most people are satisfied if they know they are buying Grade A milk, but the Grade A label may cover a multitude of sins. Of what avail is Grade A milk coming from diseased cows? Little does the lay inspector know of mastitis in cattle. Such conditions can be recognized and remedied only by the veterinarian. Within the last two months, two Oklahoma towns of county-seat size have experienced widespread epidemics of septic sore throat. After the customary tests were made on both the people and the cattle, it was disclosed that employés affected with septic sore throat had been handling the milk from public dairies. In one of these cities it was estimated that more than 50 per cent of the people were affected as a result of this epidemic. This condition occurred because of a lack of competent dairy inspection. Before this condition was straightened out, it was necessary to obtain the services of a qualified veterinarian who worked jointly with the State Board of Health. With the veterinarian's understanding of the situation and the coöperation of the physician, this matter was completely cleared up in less than 30 days.

The *National Business Magazine* tells us that 4,800,000 dairy farmers, owning 25,000,000 dairy cattle, represent one-fifth of the farm income. The economic value of this branch of the live

stock industry warrants much more consideration than heretofore has been given it by the veterinary profession. Many splendid papers on dairy inspection have been read before the American Veterinary Medical Association, but usually the time allotted did not afford an opportunity to discuss them; perhaps because we have been more interested in dividends than in investments.

It has been the writer's privilege to have had some valuable experience in dairy inspection and to have attended short courses given by the Public Health Service. At these conferences, discussions were mainly on the necessary equipment of a modern dairy. All of this is necessary in order to have sanitary surroundings, but we should not overlook in our discussions the fact that in order to obtain pure milk it must come from cows free from all disease, and it must be handled and transported in clean utensils and by people free from disease and of clean habits. The health inspection of these people should be more rigid. Only in this way can we produce milk safe for human consumption. Since 65 per cent of the goodness of the milk supply depends upon the physical condition of the cow and whether she is free from disease, with special emphasis placed upon udder disorders, it stands to reason that the veterinarian alone would be capable of directing, inspecting and checking the many deviations which make the nation's milk supply safe for human consumption.

Training for Public Health

This brings up the question of whether all veterinarians are as well qualified as they might be. Do our veterinary colleges offer a broad enough course in this subject? We must convince the medical profession and the public that the veterinarian is the best qualified person to handle this most important health measure.

Then it is up to the colleges to educate the veterinarian to the point of proficiency in sanitation and hygiene as they relate to milk-borne diseases and rural sanitation in general. Veterinarians should be able to discuss all questions pertaining to public health and be able to give well-balanced talks to civic as well as farm organizations. Also, in this connection, every veterinarian should be familiar with Title VI of the Social Security Act and fight for the recognition due our profession in this national setup for the education and control of public health.

Therefore, the next forward step for the veterinary profession to take is to complete the education of its members in this line. It should immediately undertake the standardization of

dairy inspection, keeping in mind the prevention of widespread epidemics which we have on numerous occasions experienced at a tremendous cost in money and human life; also, to do away with many foolish and ridiculous theories which this age can no longer tolerate.

Food inspection, and especially that of milk, is one of the undeveloped fields of veterinary medicine in the United States today.

Dairy Farm Inspection

At the 1927 meeting of the American Veterinary Medical Association, provision was made for the appointment of a special committee to outline a method or system of making a proper inspection of a dairy farm. This committee consisted of Drs. Louis A. Klein, Chairman, J. Payne Lowe, F. D. Holford, C. W. Eddy and J. P. Bushong. At the 1928 meeting, the report of the committee was presented and ordered printed in the JOURNAL. The report was published in the October, 1928, issue, pages 770-776.

It is of interest to record the fact that the Pennsylvania Department of Health, in the official certificate issued by veterinarians after an examination of a dairy herd has been made, provides, among other things, for a statement to the effect that the examination of the herd has been made in accordance with the standards approved by the American Veterinary Medical Association.

Midwest Veterinary Medical Conference and Clinic

The Missouri Veterinary Medical Association and the Kansas Veterinary Medical Association are sponsoring the Midwest Veterinary Medical Conference and Clinic, to be held in the Municipal Auditorium, Kansas City, Mo., June 7-8-9. The invitation to hold this event in Kansas City was extended by the Kansas City Veterinary Medical Association in November, 1937. The invitation was accepted and joint committees were appointed by the two state associations. These committees are in charge of the arrangements, and plans are being made to take care of 1,000 veterinarians and their wives from Kansas, Missouri, Nebraska, Iowa, Oklahoma, Minnesota, Arkansas, Illinois, Colorado, Kentucky and Tennessee. Drs. H. W. Young and S. J. Schilling, both of Kansas City, are General Chairman and Corresponding Secretary, respectively, of the conference.

ONE HUNDRED PER CENT MEAT INSPECTION*

By W. A. LAWRENCE, *San Antonio, Texas*

For about 40 years the federal government has maintained antemortem and postmortem meat inspection in plants doing an interstate business. Today this federal meat inspection covers only about 65 per cent of the meat and meat food products consumed in the United States, according to information revealed by a questionnaire recently sent each state. The remaining 35 per cent is prepared and processed by independent packing-plants and slaughter-houses, some with forms of local inspection, city, county, or state, which may be from good, bad or indifferent, to none at all; and all states, with the exception of one, have grossly neglected to follow the leadership of the federal government in making this 35 per cent of the meat supply of this nation safe for human consumption by knowingly permitting animals to be slaughtered for food under deplorable and insanitary conditions. The questionnaire further revealed that of this 35 per cent only about 5 per cent was under the supervision of a veterinary inspector; about 8 per cent had lay inspectors, and the great majority had no inspection whatever. In one state 95 per cent of the meat prepared for human consumption had no inspection. It is only logic that meat sold locally should have as adequate inspection as that sold interstate.

HOW SHOULD THE COST OF UNINSPECTED MEATS BE COMPUTED?

Shall the lack of adequate inspection or no inspection at all be reckoned in dollars and cents, or shall it be considered from the widespread loss of human life, brought about by consumption of unwholesome meat and meat products, treated as referred to later, by the addition of preservatives which are responsible for nephritis, gastric and duodenal ulcers, with other digestive disturbances in humankind. Society as a whole should not be willing, and would not be willing, if they knew the conditions existing in independent packing-plants and slaughter-houses, to go farther without adequate state and federal supervised meat inspection, directly affecting the 35 per cent of uninspected meats today.

"No one voluntarily accepts food of questionable cleanliness or of doubtful wholesomeness," yet there is sold and consumed meat and meat products which are from diseased animals, or

*Presented at the seventy-fourth annual meeting of the American Veterinary Medical Association, Omaha, Neb., August 16-20, 1937.

prepared with prohibited and dangerous preservatives, such as sodium sulfide, boric acid and benzoate of soda, the addition of polluted water, excessive arsenic in chili pepper, excessive and dangerous amounts of sodium nitrite and nitrate, prohibited dyes and coloring matter, larvae- and egg-infested cereals, condiments, vegetables, fruits and nuts improperly cleaned, moldy or of such quality to be questionable from a standpoint of edibility; spices that are moldy or contain quantities of stems, leaves, hulls, and other inert and foreign materials rendering such products unfit for human consumption.

An alarming feature of our present meat-packing situation is that animals, whose owners know they will not qualify for slaughter in plants having proper inspection, gravitate to houses in which inspection is lax or lacking, which permits the killing of diseased live stock for food purposes.

Public Must Be Informed

People are always in a receptive mood for anything that tends to the betterment of their living conditions if they are aware of a bad condition which may be remedied. Public sentiment will be behind any movement whereby the health of the public can be benefited. The consuming public must be advised with reference to the dangers encountered when buying meat under present conditions. We, as veterinarians, must educate the consuming public to the extent that it will demand meat which has been properly inspected and prepared under sanitary conditions. If our veterinary college curriculum does not include a complete study of this most important question of meat inspection, then it behooves our educators to see that this is done in order that veterinarians may meet the demands made by the public-health importance of proper and complete meat inspection. In the veterinary profession lies the duty of educating the people to the existing conditions and in that manner creating a demand for adequate meat inspection.

Only when there has been created a demand for properly inspected meat will there be laws passed replacing the filthy and contaminated slaughter-houses, the owners of which are pleased to term their products "home dressed meats," receiving lay inspection or no inspection, when competent modern veterinary inspection of live stock and meat for human food is available.

Meat inspection is a function of the veterinarian and it is an obligation and a duty of the veterinary organizations to advance a modern state-wide meat control service.

In the field of meat inspection there are unlimited opportunities open to the veterinarian. There is a dire need for modern, state-wide meat inspection in all packing establishments. We must fill this field with competent veterinarians and at the same time accomplish the purpose of adequate inspection.

If we are guardians of the public health, through our control and eradication of animal diseases, and our condemnation of unfit food, then it is our duty to prevent the consumption of meat and meat-food products affected with transmissible diseases, which find their way to market through these various slaughter agencies which enjoy the freedom of no inspection. It is only too apparent that the consuming public is unaware that daily there is sold through markets meat from animals affected with parasitic and infectious diseases that are transmissible from animal to man. The fact that there is more filth and disease being taken into the human system by eating uninspected meats than any other food product cannot be successfully contradicted. We, the veterinarians of this nation, have a function to perform and owe a duty to the people of this country in making this necessary food safe for human consumption.

Packers Interested

I cannot help but refer to the paper read by Oscar G. Mayer, before the 72nd annual meeting of the A. V. M. A. in Oklahoma City, "the subject of adequate inspection has been before the meat-packing industry for many years. The first official step taken was the adoption of a motion by the executive committee of the Institute of American Meat Packers in September, 1929, that a resolution be introduced in the next convention of the Institute authorizing the appointment of a commission to work out and execute plans for achieving a uniform state inspection law." The resolution, with which I am sure all of you are familiar, was presented and adopted in the same year without a dissenting vote. Yet there has been little or nothing done about this important proposal as it directly relates to the 35 per cent of the nation's meat supply which is inspected inadequately, if at all.

After consideration of the steps necessary to this goal, it appears essential to the writer that federal funds be tendered to the states through the Social Security Act, to be used in full and complete coöperation with the states, seeking adequate state and federal meat inspection control, which would forever outlaw the slimy slaughter-houses to be found in every state in the nation. Or, if this cannot be done, let an additional two million

dollars be added annually to the federal meat inspection appropriation, to be used in coöperation with the 48 states in bringing about a safe and sane meat inspection which will eliminate unhealthful, unwholesome and disease producing meat products, represented in the 35 per cent inadequately inspected.

A uniform law, patterned as closely as practical after the federal law, should be prepared and presented to all state legislatures, making it clear what the intent of such a law is, and asking that it be passed without modification. This, on the face of it, seems a hopeless task, yet it will take some kind of drastic action to bring about proper meat inspection on a nation-wide basis.

Educational Program Needed

I have in mind a plan which would provide for a nation-wide educational movement, giving the housewives, domestic science departments of high schools, home economics departments of colleges and all other institutions of higher learning, an insight into the actual conditions of the 35 per cent of uninspected meat supply of this country, made possible with federal funds through the United States Public Health Service, the Social Security Act, or the United States Department of Agriculture, by the use of slides and motion-pictures in conjunction with lectures by veterinarians who alone have first-hand information which should be given the public in the same efficient manner with which the veterinarians of the country have demonstrated their ability to control contagious and infectious diseases in animals. Why not ask the Section on Sanitary Science and Food Hygiene to go on record and ask the A. V. M. A. to appoint a committee to take this all-important matter up with the motion-picture industry, to be sponsored by federal coöperation and funds, with the hope that satisfactory arrangements could be made to bring the actual conditions before the film audience of the nation, with the further hope that the public would quickly demand a health stamp of approval on every primal part of meat sold in this country.

Through this educational program, and through this kind of a program only, can we hope to secure the backing and coöperation of civic bodies, municipal and state organizations, to the end that they will insist on their state legislatures passing the 100 per cent competent meat inspection law, and thus obtain the federal funds to be used coöperatively with the states in carrying out this program. It is my sincere hope that the Section on Sanitary Science and Food Hygiene will consider the importance of this matter and request the A. V. M. A. as a body to take some

definite action, seeking to bring about at the earliest possible date a better condition as it relates to this uninspected portion of the nation's food supply.

SUMMARY

1. Thirty-five per cent of the nation's meat supply is uninspected.
2. We should strive for 100 per cent inspection through state and federal coöperation.
3. The veterinary profession only is qualified to direct and administer inspection.
4. Slaughter-houses should be discarded at the earliest possible date.
5. A nation-wide program of education is needed and it can be had only with federal coöperation and funds.
6. The uninformed public must recognize the facts before their coöperation can be secured to demand 100 per cent inspection.

Bureau Transfers

DR. OSCAR B. PETERSON (McK. '18), from Harrisburg, Pa., to Chicago, Ill.

DR. HAL S. NEVIUS (St. Jos. '21), from Saint Louis, Mo., to New York, N. Y., on meat inspection.

DR. CESAR CLAVELL (Tex. '35), from Chicago, Ill., to San Juan, Puerto Rico, on tuberculosis eradication.

DR. ERIC H. NORDSTRUM (Iowa '29), from Chicago, Ill., to Storm Lake, Iowa, on meat inspection.

DR. GLEN H. RANDALL (K. C. V. C. '11), from Kansas City, Kan., to Moultrie, Ga., in charge of meat inspection and Packers and Stockyards Administration.

DR. JAMES M. WINGATE (A. P. I. '28), from San Juan, Puerto Rico, to Nashville, Tenn., on tuberculosis eradication.

DR. FRANCIS W. CRAWFORD (K. S. C. '23), from Sioux Falls, S. Dak., to Kansas City, Kan., on meat inspection.

DR. OTTO W. SEHER (Colo. '13), from South Saint Joseph, Mo., to Sioux City, Iowa, in charge of meat inspection.

DR. MELVIN E. HODGSON (K. S. C. '32), from Oklahoma City, Okla., to Wichita, Kan., on meat inspection.

DR. WARD T. HUFFMAN (Gr. Rap. '08), from field inspection, to poisonous plant investigation (in charge) at Salt Lake City, Utah.

DR. JOHN S. KOEN (K. C. V. C. '07), from Chicago, Ill., to Des Moines, Iowa, on hog cholera control.

DR. WILBUR MCPHERSON (O. S. U. '15), from San Juan, Puerto Rico, to Oklahoma City, Okla., on tuberculosis eradication and Bang's disease control.

DR. M. W. SCOTHORN (O. S. U. '33), from Oklahoma City, Okla., to New York, N. Y., on meat inspection.

THE EFFECTS OF THE FLUORIDES AND CHLORIDES OF SOME OF THE ALKALI ELEMENTS ON RESPIRATION AND BLOOD-PRESSURE IN THE DOG*

By D. A. GREENWOOD, E. A. HEWITT *and* V. E. NELSON

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A renewed interest has been manifest in a study of fluorine compounds recently. This interest is due largely to the observations that excessive fluorine found in phosphatic mineral supplements causes harmful effects in farm animals, and that excessive fluorine in drinking water (1 part per million or above)[1, 2] produces mottled enamel in man and other animals.

DESCRIPTION OF THE ANIMALS

These experiments were performed on dogs. Seventeen dogs, nine males and eight females, were used. The smallest weighed 6.00 kilos and the largest 26.80 kilos. The exact ages of the dogs were not determined; however, they were all mature animals. Usually, two dogs were used in the investigation of each compound. However, one dog was used in the study of sodium fluosilicate, and five dogs were employed in the study of sodium fluoride.

METHODS AND APPARATUS

The experimental methods were the same as are routinely used in pharmacological and physiological studies involving the use of a long paper kymograph, mercury manometer, pneumograph, time marker, and signal magnet. Three per cent pentobarbital-sodium (Abbott), administered either intraperitoneally or intrapleurally on the basis of 30 mg per kilo of body weight, is ordinarily employed for anesthesia. The general procedure in performing an experiment was to inject small quantities of the compound, after a preliminary tracing had been made. The preliminary tracing was assumed to be normal for the animal under anesthesia; thus the effects on the blood-pressure and respiration resulting from the injection of the salts could be evaluated. The dosage of the salt was gradually increased, until a significant change was produced on respiration and blood-pressure.

*Received for publication, October 27, 1936.

Discussion

Our present knowledge regarding the effect of the intravenous injection of fluorine compounds in mammals has been confined primarily to studies employing sodium fluoride. For that reason we deemed it advisable to investigate the effects of injections of other fluorides. It was also decided that a study of the chlorides of the alkali elements would give a basis for comparison. Tables I to IV represent typical results obtained from the intravenous injections of fluorides of sodium, potassium and ammonium, and of sodium fluosilicate. The effects of intravenous injections of sodium and potassium chloride are also discussed. Table V gives results on ammonium chloride.

The data in table I represent typical experimental values of the effect of sodium fluoride on respiration and blood-pressure. The results indicate that smaller initial doses of sodium fluoride are required to influence respiration than blood-pressure. However, as larger amounts of the compound were injected, both respiration and blood-pressure were altered very markedly. The

Table I—*Effect of sodium fluoride (¼ per cent solution) on respiration and blood-pressure (female dog, 6.0 kg).*

Time	Blood-pressure (mm of Hg)	Respirations (per Minute)	Amount Injected (gm)
9:00	145	12	
9:02	140	13	0.010
9:04	150	15	0.020
9:06	150	15	0.040
9:07	145	19	0.080
9:08	135	20	0.120
9:09	120	25	
9:10	116	30	0.160
9:12	90	42	
9:13:30	120	30	
9:15*	140	49	0.200
9:16*	104	49	
9:17	96	Ceased	
9:18†	100	Artificial respiration instituted	
9:20‡	80		
9:22	76	Artificial respiration discontinued	
9:23	66	7	
9:25§	40	Ceased	
		Total	0.630

*Dyspnea.
†Strong heart-beat.
‡Heart very weak.
§Death.

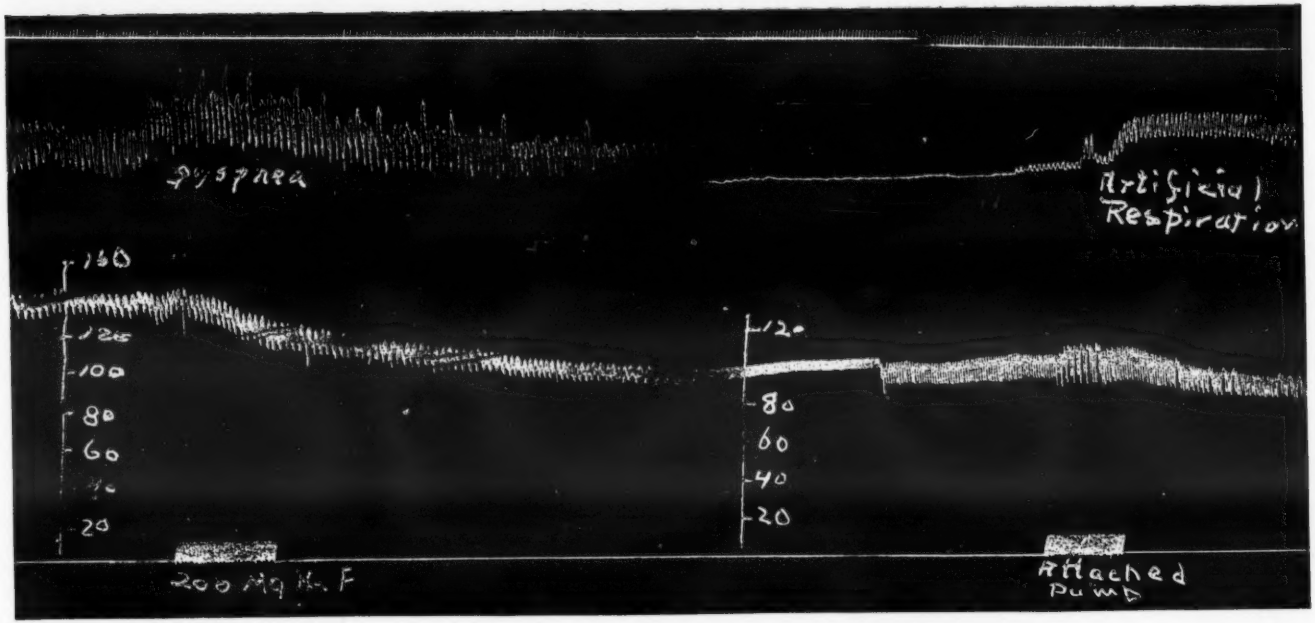

FIG. 1. The effect of sodium fluoride on respiration and blood-pressure.

increase in the respiratory rate, as well as the fall in blood-pressure, were probably due primarily to a stimulation of the respiratory, vasomotor, and cardiac centers in the medulla. Following the injection of larger doses, paralysis of these centers occurred, which likely caused death. The data in this and the other experiments reveal that respiration ceased before stoppage of the heart, which probably indicated that the respiratory center was paralyzed before the other vital centers. Hence, death probably was

TABLE II—*Effect of 2 per cent suspension of sodium fluosilicate on respiration and blood-pressure (male dog, 16.1 kg).*

TIME	BLOOD-PRESSURE (MM OF HG)	RESPIRATIONS (PER MINUTE)	AMOUNT INJECTED (GM)	REMARKS
3:32	210	15		
3:35	210	15	0.040	Temporary decline in BP to 200 mm but immediate return to 210
3:37	213	15	0.100	R. irregular
3:38	218	16		R. shallow
3:39	206	16	0.100	R. very shallow
3:40	196	20		
3:45	160	31	0.040	R. irregular and very shallow
3:48	142	43		R. irregular and very shallow
3:52	126	56		R. slightly increased in depth
4:01	120	56	0.040	
4:02	128	70		
4:03	132	77	0.100	
4:04	140	84		
4:05	143	91	0.100	R. increased in depth
4:06	146	91		
4:07	150	91	0.200	R. gradually increased in amplitude
4:08	170	91		
4:09	127	77		
4:12	154	91	0*.200	R. shallow
4:12:15	158	77		
4:14	102	91		R. markedly increased in amplitude
4:15	60	91		R. markedly increased in amplitude
4:22	60	189		R. decreased in amplitude
4:24	90	189	0.100	R. decreased in amplitude
4:25	110	189		
4:26	40	68		R. increased in amplitude R. continued for about one minute after BP dropped to minimal level
		Total	1.020	

R. = respirations.

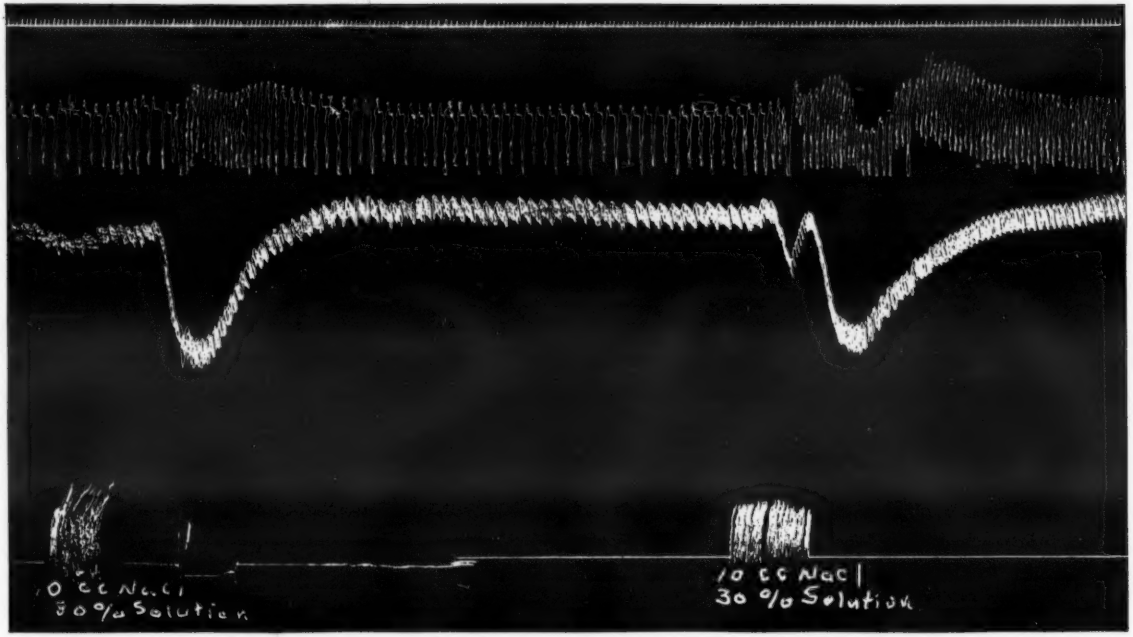

FIG. 2. The effect of sodium chloride on respiration and blood-pressure.

due essentially to respiratory paralysis. However, evidence has been presented which indicates that the sodium fluoride also exerts a specific effect on cellular respiration.

Table II summarizes the effect of sodium fluosilicate on respiration and blood-pressure. The data are somewhat similar to those obtained when sodium fluoride was injected, except that larger quantities of sodium fluosilicate were required to alter respiration and blood-pressure. Sodium fluosilicate is soluble only to the extent of 0.762 gm per 100 cc of water at 25°C., whereas 4 gm of sodium fluoride are soluble in the same volume of water at the same temperature. The solubility of the two compounds may account for the fact that larger doses of sodium fluosilicate were required to influence respiration and blood-pressure. It is interesting to note that following the administration of large doses of sodium fluosilicate respirations continued for about one minute after the blood-pressure fell to the minimal level of 40 mm of Hg, while respirations ceased before the blood-pressure fell in the case of sodium fluoride. It is also important to note that sodium fluosilicate is more toxic per kilo of body weight than sodium fluoride. The increased toxicity of sodium fluosilicate could not be correlated with its solubility.

The effects of sodium chloride are recorded in tabular form in a previous paper.[3] Initial doses of sodium chloride caused a temporary rise in blood-pressure. As larger doses were administered, a temporary rise occurred, followed by a significant drop in blood-pressure. Small doses of sodium chloride had relatively little effect on the respiratory rate. However, as larger amounts were injected, the respiratory rate increased markedly. A 30 per cent solution of sodium chloride was used because of the lower toxicity of this compound. The strongly hypertonic solution of sodium chloride, caused, at the beginning of the experiment, a stimulation of the respiratory, cardio-inhibitory and vasomotor centers in the medulla; which would be the most significant factor in increasing the respiratory rate and also cause the rise and fall in blood-pressure. The injection of larger amounts of strongly hypertonic solution would also cause an increase in blood-volume, which would tend to increase the blood-pressure. Paralysis of the vital medullary centers occurred after frequent injections of large doses of the compound. Respirations ceased before the heart stopped. Edema and hemorrhage of the lungs were observed during the latter part of the experiment. An autopsy revealed profuse edema and hemorrhage of the lungs and liver, which can be explained by assuming that the salt left the circula-

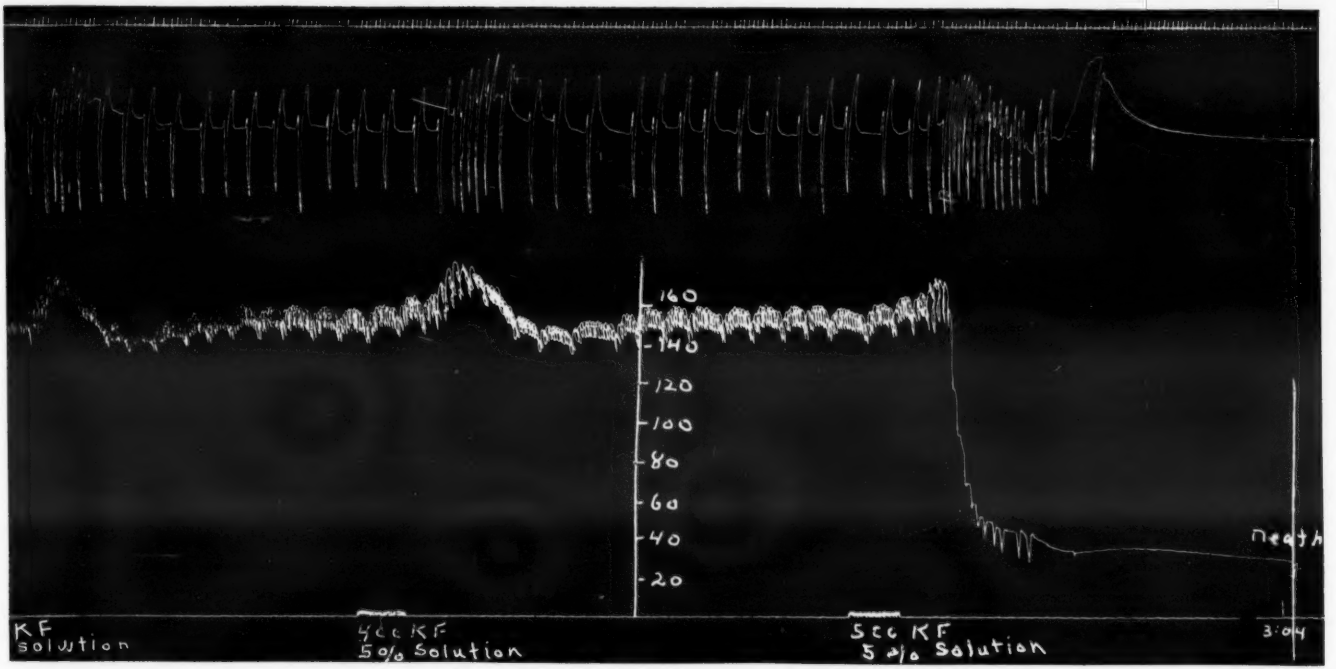

FIG. 3. The effect of potassium fluoride on respiration and blood-pressure.

tion, escaping into the tissue spaces; and this created a hypertonic tissue fluid which, by its osmotic pressure effects, drew more water into the tissue spaces.

Table III summarizes the effect of potassium fluoride on respiration and blood-pressure. The data indicate that initial small doses cause a sudden temporary increase in the respiratory rate as well as a temporary increase in blood-pressure. Respirations were influenced a few seconds before the blood-pressure increased. However, respirations persisted for about one minute after the heart ceased, and the blood-pressure fell to a minimal level. After the seventh injection, the blood pressure increased slightly, and then a precipitous fall occurred. A heart-block probably caused the death of the animal.

The effects of potassium chloride on respiration and blood-pressure are recorded in tabular form in a previous paper.[3] There is a striking similarity in the data obtained from the injection of potassium chloride and potassium fluoride. Small doses

TABLE III—*Effect of potassium fluoride (5 per cent solution) on respiration and blood-pressure (female dog, 10 kg).*

TIME	BLOOD-PRESSURE (MM OF HG)	RESPIRATIONS (PER MINUTE)	AMOUNT INJECTED (GM)	REMARKS
8:47	170	14		
8:51	162	14	0.075	R. influenced 15 sec. before BP
8:51:30	186	42		
8:52	176	17	0.075	R. influenced 15 sec. before BP
8:52:30	180	17		
8:53	176	17	0.100	R. influenced 15 sec. before BP
8:53:30	220	42		
8:54	194	19		
8:55	180	14	0.100	R. irregular
8:55:30	192	42		
8:56	180	14		
8:57	176	14	0.150	
8:57:15	191	42		R. influenced before BP
8:58	176	14		
8:59	176	14	0.150	
8:59:15	194	49		R. influenced before BP
9:02	181	16	0.200	
9:02:15	196	49		R. influenced before BP
9:02:30	40	9		R. very irregular R. persisted for 1 min. after drop of BP and then ceased Death
		Total	0.850	

R. = respirations.

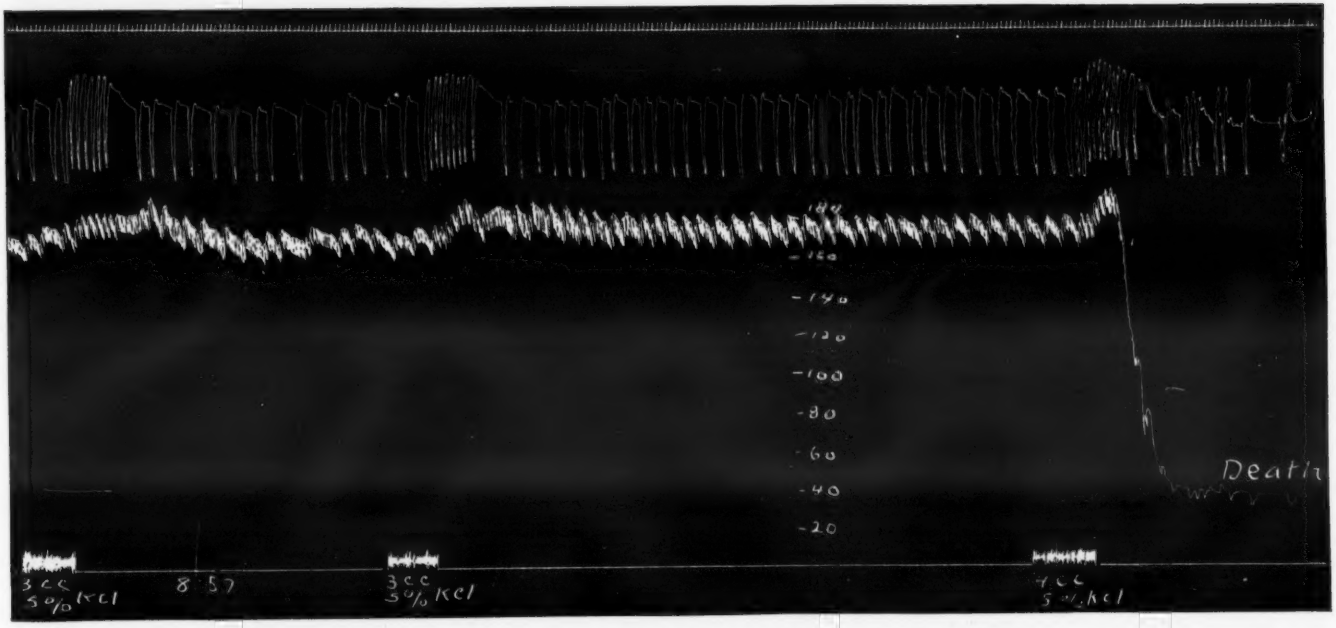

FIG. 4. The effect of potassium chloride on respiration and blood-pressure.

of potassium chloride caused a rise of blood-pressure. Following the third injection, which totaled 0.250 gm, the blood-pressure rose to 218 mm of Hg. The lethal amount was 0.850 gm, which temporarily raised the blood-pressure from 180 to 192 mm of Hg and increased the respiratory rate from 17 to 49. However this was followed in 15 seconds by a rapid fall in blood-pressure to the minimal level of 40 mm of Hg and cessation of respirations with the death of the animal. A postmortem examination of the animal showed the heart to be in a relaxed or dilated state, indicating that it had stopped in diastole (potassium inhibition).

Table IV contains a summary of the effect of ammonium fluoride on respiration and blood-pressure. The results indicate that small initial doses of the compound cause a temporary decrease in the respiratory rate and a slight fall in blood-pressure. However, as a larger dose was administered, the respiratory rate decreased temporarily, and then the rate increased markedly for a short interval of time. Following the increase in respiratory rate, respirations decreased at a very rapid rate and ceased. Blood-pressure followed the same general trend as respiration after the injection of a larger dose of the compound. It showed a temporary fall immediately following the injection and then a gradual rise to a high level, followed by a precipitous fall. The results indicate that ammonium fluoride produced a pronounced

TABLE IV—*Effect of ammonium fluoride (5 per cent solution) on respiration and blood-pressure (male dog, 6.82 kg).*

TIME	BLOOD-PRESSURE (MM OF HG)	RESPIRATIONS (PER MINUTE)	AMOUNT INJECTED (GM)	REMARKS
10:13	147	70	0.050	
10:16	145	70	0.100	
10:16:30	116	56		R. increased in amplitude
10:17	150	63		
10:18	148	63	0.150	
10:18:15	120	56		R. markedly increased in amplitude
10:19	84	56		
10:20	70	84		
10:20:30	158	83		R. decreased in amplitude
10:21	30	63		
10:23	18	Ceased		
		Total	0.300	

R. = respirations.

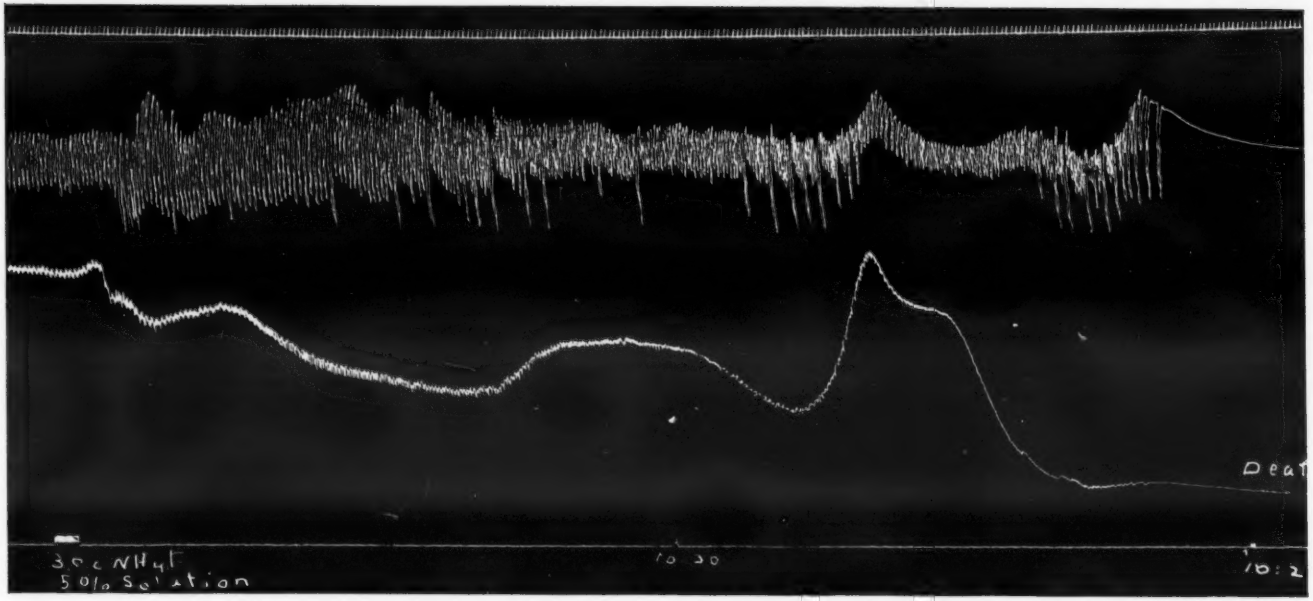

FIG. 5. The effect of ammonium fluoride on respiration and blood-pressure.

TABLE V—*Effect of ammonium chloride (5 per cent solution) on respiration and blood-pressure (female dog, 9.3 kg).*

TIME	BLOOD-PRESSURE (MM OF HG)	RESPIRATIONS (PER MINUTE)	AMOUNT INJECTED (GM)
2:04:30	165	54	0.010
2:05*	150	60	
2:05:10	165	54	
2:08	163	90	0.020
2:08:20	150	84	
2:08:30*	165	90	
2:10	164	48	0.030
2:10:20	150	48	
2:10:30†	166	48	
2:13	166	48	0.040
2:13:20	145	48	
2:13:30	166	36	
2:17	166	36	0.050
2:17:20	146	36	
2:17:30	170	36	
2:21	170	78	0.050
2:21:20	142	60	
2:27	172	72	0.100
2:27:20	146	96	
2:27:30	184	60	
2:39	180	60	0.100
2:39:20	150	84	
2:39:30	183	84	
2:49	184	114	0.150
2:49:20	154	84	
2:50	203	84	
2:58‡			
3:06	180	120	0.200
3:06:20	152	120	
3:07	216	66	
3:09	170	96	0.250
3:09:20	152	114	
3:10	185	90	
3:14	180	90	0.400
3:14:20	150	96	
3:15	200	54	
3:16	180	108	0.500
3:16:20	164	96	
3:21	170	48	0.750
3:21:20	155	78	
3:22	190	42	
3:23	188	48	0.750
3:23:20	170	60	
3:29§	170	36	1.000
3:33§	102	30	
3:34	30	Ceased	
		Total	4.400

*Gasp 30 seconds after injection.
†Gasp 30 seconds after injection. Later, gasps were more frequent.
‡Urination.
§Pulse pressure 30 mm of Hg.

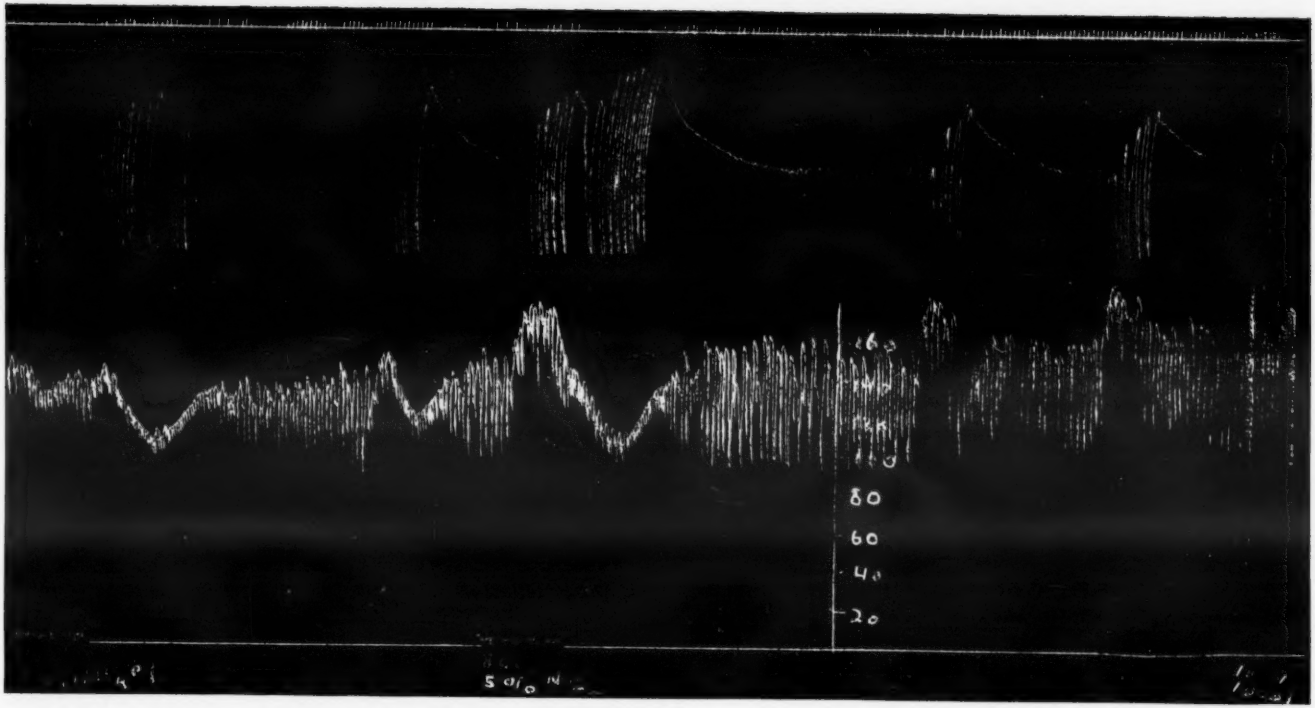

FIG. 6. The effect of ammonium chloride on respiration and blood-pressure.

stimulation and subsequent paralysis of the respiratory, vasomotor and cardio-inhibitory centers in the brain.

The effect of ammonium chloride on respiration and blood-pressure is given in table V. The general trend of the data is similar to that obtained with ammonium fluoride. However, ammonium fluoride is more toxic.

Table VI summarizes the effects of the various fluorides and chlorides on respiration and blood-pressure and also gives the lethal intravenous dosage. The data on respiration reveal in general that smaller quantities of fluorides are required to produce significant changes in respiration than chlorides of the same element or radical. The fluorides, as well as the chlorides, cause an increase in the respiratory rate. The initial increase in the respiratory rate is probably due primarily to a stimulation of the medullary respiratory center. As larger quantities of the compounds were administered, stimulation of other medullary centers occurred temporarily, followed by a partial and increasing paralysis of these centers, which finally caused death. When the fluorides are arranged in the order of minimum dosage required to produce a significant change in respiration, the following order exists: potassium fluoride, ammonium fluoride, sodium fluoride and sodium fluosilicate. When the chlorides are arranged on the same basis, the following order was observed: ammonium chloride, potassium chloride and sodium chloride.

The results on blood-pressure reveal that initial small doses of the fluorides, except potassium, produced a decline in blood-pressure; while initial small doses of the chlorides, except ammonium, caused an increase in blood-pressure. The lowering of blood-pressure is probably due primarily to a specific effect of the fluoride anion. However, the ammonium ion also produces a lowering of blood-pressure.

The data in table V reveal that there is a considerable variation in the toxicity of the fluorides and chlorides. The fluorides in general are decidedly more toxic than the corresponding chlorides. When the fluorides are listed in order of toxicity per kilogram of body weight, the following sequence was noted: ammonium fluoride, potassium fluoride, sodium fluosilicate and sodium fluoride. The data also indicate that the cations are responsible to a large extent for the toxicity of the compounds. The order of toxicity of the chlorides was: potassium, ammonium and sodium.

TABLE VI—*Effect of intravenous injection of fluorides and chlorides of some of the alkali elements.*

COMPOUND	RESPIRATIONS PER MINUTE BEFORE INJECTION OF COMPOUND	GRAMS OF COMPOUND PER KILO BODY WEIGHT REQUIRED TO PRODUCE SIGNIFICANT CHANGE IN RESPIRATION	RESPIRATIONS PER MINUTE AFTER INJECTION OF COMPOUND	BLOOD PRESSURE IN MM OF HG BEFORE INJECTION OF COMPOUND	GRAMS OF COMPOUND PER KILO BODY WEIGHT REQUIRED TO PRODUCE SIGNIFICANT CHANGE IN BLOOD-PRESSURE	BLOOD-PRESSURE IN MM OF HG AFTER INJECTION OF COMPOUND	GRAMS OF COMPOUND PER KILO BODY WEIGHT WHICH CAUSED DEATH	GRAMS F OR Cl PER KILO BODY WEIGHT PRESENT IN COMPOUND WHICH CAUSED DEATH	GRAMS OF CATION PER KILO BODY WEIGHT IN COMPOUND WHICH CAUSED DEATH
NaF	15.3	0.0064	22.6	170	0.0509	136	0.1044	0.0470	0.0574
Na_2SiF_6	15.0	0.0150	31.0	210	0.0150	160	0.0638	0.0387	0.0156
NaCl	20.0	0.1166	25.0	150	0.0165	170	3.3333	2.0223	1.3110
KF	20.0	0.0038	24.0	152	0.0055	166	0.0737	0.0241	0.0496
KCl	14.0	0.0075	36.0	164	0.0075	182	0.0850	0.0393	0.0457
*NH_4F	38.0	0.0053	70.0	175	0.0126	141	0.0413	0.0212	0.0201
NH_4Cl	54.0	0.0032	90.0	165	0.0012	150	0.5048	0.3348	0.1700

*One of the animals received a small quantity of ether before fluorine compound was injected.

SUMMARY

1. Smaller quantities of fluorides are required to produce significant changes in respiration than chlorides of the same element or radical.

2. In the order of minimum dosage required to produce a significant change in respiration, the following sequence was observed: potassium fluoride, ammonium fluoride, sodium fluoride and sodum fluosilicate. The chlorides, on the same basis, arranged themselves as follows: ammonium chloride, potassium chloride and sodium chloride.

3. Initial small doses of the fluorides, except potassium fluoride, cause a decrease in blood-pressure; whereas, initial small doses of the chlorides, with the exception of ammonium chloride, cause an increase in blood-pressure. Administration of larger doses of all of the compounds produced a very low blood-pressure.

4. In the order of minimum dosage required to produce a significant change in blood-pressure, the fluorides can be arranged as follows: potassium fluoride, ammonium fluoride, sodium fluosilicate and sodium fluoride.

5. The fluorides were considerably more toxic than the chlorides. When the fluorides are listed in the order of toxicity per kilogram of body weight, they may be arranged as follows: ammonium fluoride, potassium fluoride, sodium fluosilicate and sodium fluoride.

6. The order of toxicity of the chlorides was as follows: potassium chloride, ammonium chloride and sodium chloride.

7. The cations are responsible to a large extent for the difference in toxicity of the various compounds of fluorine and chlorine.

8. The method and rate of injection are important factors in determining the lethal dosage of a compound.

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A comfortable house is a great source of happiness. It ranks immediately after health and a good conscience.

—*Sydney Smith*

NOTE ON THE USE OF SULFANILAMIDE IN THE TREATMENT OF CHRONIC BOVINE MASTITIS DUE TO STREPTOCOCCI*

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Many reports have appeared in the medical literature of the United States and other countries in the last two years on the use of para-aminobenzenesulfonamide and its derivatives in the treatment of various infectious diseases in man and small laboratory animals. Diseases in man caused by hemolytic streptococci of human origin were first found to respond favorably to the therapeutic action of the drug and since then it has been used with a fair amount of success in several other bacterial infections. On the other hand, litle information has appeared in the veterinary literature of the world concerning the use of para-aminobenzenesulfonamide in the treatment of diseases of domestic animals in which bacteria are the etiological agents. It would appear that there might be several diseases in this field which should respond favorably to such therapy.

Chronic bovine mastitis caused by streptococci has been considered in this connection. So far, however, only one report[1] has been observed in which this drug or one of its derivatives has been used in the treatment of the disease. In this case, a cow, which had recently freshened, with clinical mastitis and abnormal udder secretion containing long-chain streptococci, was treated with six 75-grain doses of prontylin. Prontylin is the soluble form of para-aminobenzenesulfonamide. Fifteen 5-grain tablets were dissolved in water and the solution given as a drench twice a day for three days. After four doses had been given, no streptococci could be found either in smears of the secretion or on culture. Two more doses were given and at the end of two months the animal was still free of these organisms. The author stated further that two more cases of mastitis in which long-chain streptococci were present had been reported as having been successfully treated with prontylin. It cannot be determined from this report what types of streptococci were involved in these cases.

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In view of the favorable results obtained with one of the derivatives of para-aminobenzenesulfonamide, it appeared desirable to investigate the possible therapeutic value of the uncombined drug, sulfanilamide, on lactating cows with chronic streptococcic mastitis. The animals used for the experiment were part of a small dairy herd which was known to have had a moderate amount of such infection for several years. Of the 32 milking cows in the herd, six were found to be carrying streptococci in one or more quarters by means of the Hotis test[2] and microscopic examination of smears of incubated milk. Before treatment was undertaken, milk samples were taken from each quarter of these six animals to find out which ones were infected. However, the milk from all of the infected quarters appeared to be normal on gross inspection and although a certain amount of induration was present in each quarter the changes were of a limited nature. In addition to these two tests, the milk was cultured on 5 per cent horse-blood-agar plates. When streptococci were found on these plates, the colonies were picked for identification in differential culture media. The media used for this purpose has been described in a previous publication.[3] This same procedure was used in subsequent examinations of the milk to ascertain the results of the treatment and to identify any strains of streptococci still present in the infected quarters.

In the course of this work two experiments were carried out. In the first one, the dosage of sulfanilamide (Merck) was the same as that used in the experiment cited.[1] The method of administration, however, differed in that the drug was given on the feed in the form of a powder instead of as a drench and also the drug differed slightly in composition from prontylin. The course of treatment consisted of 75 grains of sulfanilamide sprinkled on the grain at the morning and evening feedings for three days, making six doses in all or a total of 450 grains for each individual. The animals ate the medicated feed without hesitation. Treatment was started in the afternoon of the first day, continued twice a day on the two following days, and the final dose was given on the morning of the fourth day.

Following the termination of the first experiment, it appeared desirable to investigate the possibilities of the drug further by using somewhat larger doses over a longer period of time. Accordingly, arrangements were made to treat four animals in the same herd which were carrying streptococci in one or more quarters with ½ ounce of sulfanilamide twice a day for eight days. The first dose was given in the afternoon of the first day

and the last dose on the morning of the ninth day. The larger amount of the drug was sprinkled on the feed in the dry state, as in the first experiment, and it was eaten just as readily as the smaller dose. Inasmuch as the cows on which the remedy was to be tested were valuable purebred animals, it appeared advisable to test the action of the proposed dosage of sulfanilamide on a less valuable lactating cow before undertaking the second experiment. Sixteen ½-ounce doses were given in eight days and no unfavorable effects, except a slight diarrhea, were observed in the animal over a period of several weeks. Similarly, the drug appeared to be entirely innocuous for the four animals which were treated later, as far as health and milk production were concerned, and, in addition, no diarrhea was observed. Milk samples were collected from these cows as in the previous experiment, before treatment was started, during the feeding period and for several weeks after administration of the drug was discontinued.

The results of the first experiment are shown in table I. The first milk samples were collected (August 23, 1937) immediately before the first dose of sulfanilamide was given in the afternoon. The last dose was given on the morning of the fourth day and the milk samples drawn in the afternoon. It is apparent that this dosage of sulfanilamide had no effect on the course of the infection in these cows, since all of the quarters harboring streptococci prior to treatment continued to carry them for at least several weeks after the medication had been discontinued.

The numbers in the column under the Hotis test (H) show that in four cows *Streptococcus agalactiae* (culture 1 in table III) was present in all of the infected quarters. The strains of streptococci designated *S. agalactiae* are atypical insofar as their action on trehalose (table III) is concerned. Ordinarily this substance is fermented by *S. agalactiae*. However, some of these strains did ferment trehalose when tested some time later after being held for a time on artificial media. It would seem, therefore, that they may be properly designated as *S. agalactiae*. In the two remaining animals (03 and 44) the strains of streptococci were similar to types found somewhat less frequently than *S. agalactiae* in streptococcic mastitis. Consequently it would appear that the drug is not effective against either variety.

When it became evident that sulfanilamide was of no value in the amounts used in this experiment, it was decided to increase the dosage and continue its administration over a longer period. The cows used in this experiment included three animals (17, 36 and 03) which had been used in the previous work and one (75)

TABLE I—*First experiment. Results of bacteriological examination of quarter samples.*

COW	QUARTER	EXAM.	8-23-37	8-26-37	8-30-37	9-2-37	9-9-37
17	R. R.	H	−	−	−	+	−
		S	−	−	−	+	−
	L. R.	H	+1	+1	+	+	+
		S	+	+	+	+	+
	R. F.	H	+1	+1	+	+	+
		S	+	+	+	+	+
	L. F.	H	+1	+1	+	+	+
		S	+	+	+	+	+
46	R. R.	H	−	−	−	−	−
		S	−	−	−	−	−
	L. R.	H	−	−	−	−	−
		S	−	−	−	−	−
	R. F.	H	+1	[redacted] +1	[redacted] + [redacted]	+	+
		S	+	+	+	+	+
	L. F.	H	+1	+1	+	+	+
		S	+	+	+	+	+
18	R. R.	H	−	−	−	−	−
		S	−	−	−	−	−
	L. R.	H	+1	+1	+	+	+
		S	+	+	+	+	+
	R. F.	H	−	−	−	−	−
		S	−	−	−	−	−
	L. F.	H	[redacted] +1	+1	+	+	+
		S	+	+	+	+	+
03	R. R.	H	−	−	−	−	−
		S	−	−	−	−	−
	L. R.	H	−	−	−	−	−
		S	−	−	−	−	−
	R. F.	H	−2	−	−3	−	−
		S	+	−	+	+	+
	L. F.	H	−	−	−	−	−
		S	−	−	−	−	−

(Concluded on page 552)

TABLE I—*First experiment. Results of bacteriological examination of quarter samples. (Concluded)*

Cow	Quarter	Exam.	8-23-37	8-26-37	8-30-37	9-2-37	9-9-37
36	R. R.	H	−	−	−	−	−
		S	−	−	−	−	−
	L. R.	H	−	−	−	−	−
		S	−	−	−	−	−
	R. F.	H	+	+1	+	+	+
		S	+	+	+	+	+
	L. F.	H	+1	+1	+	+	+
		S	+	+	+	+	+
44	R. R.	H	+3	+3	+3	+	+
		S	+	−	+	+	+
	L. R.	H	−	−	−	−	−
		S	−	−	−	−	−
	R. F.	H	−	−	−	−	−
		S	−	−	−	−	−
	L. F.	H	−	−	−4	−	−
		S	−	−	+	−	−

H = Hotis test.
S = Microscopic examination of smears of incubated milk.
+ = Streptococci present.
− = Streptococci absent.
R.R. = Right rear quarter.
L.R. = Left rear quarter.
R.F. = Right front quarter.
L.F. = Left front quarter.
Numbers 1 to 4 refer to reactions of streptococci shown in table III.

which had freshened after the initial survey and was found to be carrying *S. agalactiae* in all four quarters. Milk samples were taken in the afternoon (September 13, 1937), as in the previous experiment, and the first dose of the drug given at that time. This was 18 days after termination of the first course of treatment. The last dose was given on the morning of the ninth day (September 21, 1937) and a milk sample taken in the afternoon.

The results of this experiment (table III) indicate that sulfanilamide in the doses used not only has no therapeutic effect on the streptococci responsible for this type of bovine mastitis but it apparently is ineffective in preventing the spread of the infection to other quarters of the same animal. This is shown by the fact that *S. agalactiae* appeared in the right rear quarter of cow 36 three days after treatment with the larger dose of the drug had been started. Subsequent examinations of the milk seemed to indicate that the infection had become definitely established.

TABLE II—*Second experiment. Result of bacteriological examination of quarter samples.*

COW	QUARTER	EXAM.	9–13–37	9–16–37	9–21–37	9–23–37	9–30–37	10–11–37
17	R. R.	H	−	−	−	−	−	−
		S	−	−	−	−	−	−
	L. R.	H	+1	+	+	+	+	+1
		S	+	+	+	+	+	+
	R. F.	H	+1	−	−	+	+	+1
		S	+	−*	+	+	+	+
	L. F.	H	+1	+	+	+	+	+1
		S	+	+	+	+	+	+
36	R. R.	H	−	±	+	+	+	+1
		S	−	+	+	+	+	+
	L. R.	H	−	−	−	−	−	−
		S	−	−	−	−	−	−
	R. F.	H	+1	+	+	+	+	+1
		S	+	+	+	+	+	+
	L. F.	H	+1	+	+	+	+	+1
		S	+	+	+	+	+	+
03	R. R.	H	−	−	−	−	−	−
		S	−	−	−	−	−	−
	L. R.	H	−	−	−	−	−	−
		S	−	−	−	−	−	−
	R. F.	H	±5	−	−	+	−	+6
		S	+	+	+	+	+	+
	L. F.	H	−	−	−	−	−	−
		S	−	−	−	−	−	−
75	R. R.	H	+1	+	+	+	+	+1
		S	+	+	+	+	+	+
	L. R.	H	+1	−	+	+	+	+1
		S	+	+	+	+	+	+
	R. F.	H	+1	+	+	+	+	+1
		S	+	+	+	+	+	+
	L. F.	H	+1	+	+	+	+	+1
		S	+	+	+	+	+	+

*Streptococci on blood-agar plates.
H = Hotis test.
S = Microscopic examination of smears of incubated milk.
+ = Streptococci present.
− = Streptococci absent.
± = Suspicious reaction.
R.R. = Right rear quarter.
L.R. = Left rear quarter.
R.F. = Right front quarter.
L.F. = Left front quarter.
Numbers 1, 5 and 6 refer to reactions of streptococci in table III.

TABLE III—*Cultural reactions of streptococci.*

CULTURE	HEMOLYSIS (BLOOD-AGAR PLATE)	LITMUS MILK			METH. BLUE MILK		LACTOSE	SALICIN	MANNITE	TREHALOSE	SORBITOL	AESCULIN	SODIUM HIPPURATE
		A	C	R	C	R							
1*	Weak	+	+	S	−	−	+	+	−	−	−	−	+
2	Weak	+	+	S	+	P	+	+	−	+	+	−	+
3	Weak	+	−	−	P	S	+	+	−	+	+	−	+
4	Weak	+	+	P	+	+	+	+	−	+	−	+	+
5	Weak	+	+	S	−	−	+	+	−	+	+	+	+
6	Weak	+	−	−	+	P	+	+	+	+	+	+	+

**S. agalactiae.*
A = Acid.
C = Coagulation.
R = Reduction.
\+ = Complete.
P = Partial.
S = Slight.

Cow 03 was included in the group because of the fact that the streptococci in the right front quarter differed in their cultural reaction on differential media from *S. agalactiae*. It can be seen in table III that the reactions of the strains isolated from this quarter on the two occasions were not exactly the same but the difference was not marked. The cause of this variation is not well understood. The results obtained in this experiment are in agreement with reports which have been received informally from time to time to the effect that sulfanilamide has relatively little value in the treatment of chronic bovine mastitis in which streptococci are involved.

Since the conclusion of this experiment, Allott[4] has reported on the use of sulfanilamide in cows with severe cases of streptococcus mastitis. Thirty grams of the drug were administered in capsules twice a day for periods of 12 to 14 days. It was concluded from the results that some beneficial therapeutic effect was produced, but a permanent cure was not attained.

SUMMARY

Two experiments are reported on the use of sulfanilamide (para-aminobenzenesulfonamide) in the treatment of chronic streptococcic mastitis in dairy cattle. Six cows carrying mastitis streptococci in one or more quarters were treated twice daily for three days with 75-grain doses of the powdered drug sprinkled on the feed. The milk was examined bacteriologically for streptococci before and after treatment. No curative effect on the disease was observed.

In a second experiment ½ ounce of sulfanilamide in powder form was sprinkled on the feed of four cows twice a day for a

period of eight days. The milk was examined bacteriologically on several occasions during and after treatment. The result of this work was the same as found in the first experiment.

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Twelfth International Veterinary Congress Prize

One of the features of the New York meeting will be the award of the Twelfth International Veterinary Congress Prize. This will go to the member of the American Veterinary Medical Association, who, in the opinion of the members of the committee, has made the most noteworthy contribution to the advancement of veterinary science during the year—the interval between the 1937 and 1938 meetings. The award will be in cash and will consist of the interest on a $5,000.00 endowment created in 1926, on the recommendation made by the Finance Committee of the Twelfth International Veterinary Congress. The amount of the award this year will be about $137.50.

Selection of the 1938 winner will be made by a committee consisting of Drs. Oscar V. Brumley, *Chairman;* John R. Mohler, Harry W. Jakeman and W. H. Feldman and Col. Robert J. Foster. The committee will appreciate hearing from any members of the A. V. M. A. who desire to offer any suggestions, recommendations or data that will be of assistance in selecting the 1938 recipient of the award. Communications should be addressed to Dr. Brumley.

Phi Zeta at Cornell Initiates

At a recent meeting of Alpha Chapter of Phi Zeta, Honorary Society in Veterinary Medicine, the following students of New York State Veterinary College at Cornell University were elected to active membership:

Seniors: J. W. Earl, H. K. Cooper and M. H. Covert.

Juniors: W. B. Bell, I. I. Sprecher, E. J. Sunderville and R. Ferber.

Faculty: Drs. C. W. Barber and H. T. Batt.

At the same meeting, Dr. L. E. Moore, of Amsterdam, N. Y., was elected to honorary membership. H. L. G.

PROLAPSE OF THE UTERUS IN THE MARE*

By CHAS. HAASJES, *Shelby, Mich.*

Writers of veterinary text-books as well as many veterinary practitioners have always considered a complete prolapse of the uterus in a mare as usually being fatal to the animal. However, during the 18 years that I have been in practice, it would appear that luck and I have worked together because I do not believe that there is anything in the whole category of veterinary practice in which I have been as fortunate in handling as this condition.

I freely admit that I have no special method for handling eversions except as a routine procedure. In each of the three cases which I am reporting, there was a complete prolapse and each mare made a complete recovery. Not one of them was a very valuable animal.

In the first case, the organ was reduced by the same technic that I use in the cow. The vulva was sutured and, on account of an unfavorable prognosis, the owner was instructed how to feed the colt by hand. I also told him to let me know when the mare died so that I would not make an unnecessary call to remove the sutures. As I did not hear from him, I made the call. Upon my arrival at the farm, the owner came out of the house and asked when he could put the mare to work. When I entered the barn to look at her, I noticed that she was eating a hearty meal.

The second case was also a complete prolapse, but the fetus, a fully mature mule colt, was dead. I used the same technic in this case and again I gave an unfavorable prognosis. The owner said that he could remove the sutures himself. A few days later, the owner called me and said, "The next time you tell me something, I am going to believe just the opposite. The mare is fine except that her udder is swollen and caked." I instructed him how to milk the mare and gave directions for reducing the milk-flow. The mare got along nicely.

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The third case was a mare in a large, rough, hilly pasture. The mare objected to the uterus hanging against her hocks and she tried to kick it like a wild vicious mule. From the actions of the mare, the uterus would be thrown up into the air and land somewhere above the base of her tail and then come down with a snap against her hocks, which would only make her kick some more. Meanwhile, the owner, a rather penurious individual, bewailed the fact that he had called me and would have to pay me a fee in addition to losing the mare. We caught her and replaced the uterus. We were a long distance from civilization, with no clean water available, but the vulva was sutured as in the other cases. The mare was tied in such a position that the hind parts were higher than the fore part of the body, and much to my surprise she recovered.

In none of these cases was medicine of any kind introduced into the uterus or given to the mare. It would have been advisable, however, to have given a prophylactic dose of tetanus antitoxin.

ANOPLOCEPHALIASIS IN MINNESOTA HORSES*

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A five-month-old female colt was received recently at the Veterinary Division from a farm in Pine County. Through the courtesy of Dr. W. L. Boyd, an examination was made for intestinal parasites. Twelve large specimens of *Anoplocephala magna* were collected from the small intestine.

Tapeworms have not been encountered frequently in horses examined postmortem or from fecal samples in Minnesota. During the past 20 years, five cases of anoplocephaliasis appear in the records of the Veterinary Division and of the Department of Entomology and Economic Zoölogy. Of these infections, two were the large tapeworm, *Anoplocephala magna*, and three were the small tapeworm, *Paranoplocephala mamilliana*. Forty-two specimens of the latter species were collected in one instance. The perfoliate tapeworm, *Anoplocephala perfoliata*, has not been taken.

*Paper No. 1586 of the Scientific Journal Series of the Minnesota Agricultural Experiment Station, Saint Paul. Received for publication, January 27, 1938.

Hall and Hoskins,[1] after having reviewed the literature on tapeworms of horses in the United States, concluded that *A. magna* is the commonest form. They noted that it frequently occurs in enormous quantities. These authors reported *Paranoplocephala mamilliana (Anoplocephala mamilliana)* for the first time and stated that *Anoplocephala perfoliata* had never been reported, correctly, from equines in the United States.

Crocker[2] reported ten cases of *A. magna*, four of *A. perfoliata*, and none of *P. mamilliana* from horses autopsied at the University of Pennsylvania Veterinary School during the period from 1910-1919.

Davis[3] collected what he thought to be *A. perfoliata* in great numbers from a horse in Ontario, Canada.

Two species of tapeworms, *A. magna* and *A. mamilliana*, are recorded[4] as common in Montana horses.

Schwartz, Imes and Wright[5] stated that all three species of tapeworms are found in American horses but that *A. perfoliata* occurs much less commonly than the other two species.

Baker and Fincher[6] reported a case from New York in which a mare was infested with 19 specimens of *A. magna*, eight in the small intestine and eleven in the large intestine.

The life cycle of the members of the large family of anoplocephaline tapeworms, which include, in addition to those of the horse, *Moniezia* from sheep and *Cittotaenia* from rabbits, as common forms, has not been completely demonstrated experimentally. Opinion has been divided among workers as to the nature of the cycle. Some believed it to be direct while others thought an intermediate host to be involved. Stunkard,[7] following a review of the life history studies on the anoplocephaline cestodes, carried out a long series of experiments to test the theory of direct transmission of *Cittotaenia*. The experimental rabbits remained uninfected even though attempts were made to simulate the conditions to which the eggs would be exposed in nature. Following these experimental studies, he concluded that the "onchospheres are not infective for the primary host; that an intermediate host is required; that the onchospheres when voided are infective for the intermediate host; and that the larvae develop into the cysticercoid stage in the body cavity of some invertebrate animal."

Stoll[8, 9] following extensive studies on the epidemiology of *Moniezia expansa* infections in sheep, was led to believe the life cycle is direct, the sheep acquiring infection through ingestion of the infective eggs. Stunkard[10, 11] in continuing his search for a suitable intermediate host, fed eggs of *Moniezia expansa* to species of free-living mites (*Galumna* sp.) and obtained in the

body cavity of the intermediate host, for the first time, a series of developmental stages ranging from onchospheres to cysticercoids. A lamb fed mites previously exposed to *Moniezia* eggs became infected. While these findings tend to substantiate Stunkard's[7] conclusions that the life cycle is completed through the mediary of an intermediate host, controlled feeding experiments are necessary to verify them.

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CYSTIC CALCULUS IN A DOG*

By HILTON A. SMITH, *Pullman, Wash.*
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The accompanying photograph is of a calculus in the bladder of a dog, possibly of interest because of its unusual size. The stone was of the calcium oxalate type, slightly yellowish in color, hard, dense, and very rough. It was covered everywhere with little protuberances half the size of a grain of wheat, which were almost always rounded rather than jagged. We are told[1] that stones of this type usually have their surfaces smoothed out by later deposition of carbonates and phosphates, which fill the depressions to the level of the protuberances, but this had not occurred in the present instance. When examined, the size of the calculus, almost two inches in its greatest diameter, was just sufficient to fill the cavity of the bladder, but the latter was undoubtedly capable of greater expansion.

The bladder wall everywhere presented the picture of a chronic ulcerative cystitis: a roughened and eroded, intensely reddened, almost bleeding mucosa; the whole wall considerably thickened

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and indurated. These changes did not extend appreciably into the ureters or urethra. This fact, together with the absence of any discernible pus, would suggest that secondary bacterial infection, a common finding in cystolithiasis, had assumed little or no importance in this instance. No other calculi were found in any part of the urinary tract. Other organs were approximately normal.

FIG. 1. Bladder of dog showing calculus *in situ*.

Owing to the fact that the animal was presented for destruction, little exact clinical information is available. The dog was a female of mongrel type, weighing around 25 pounds, and estimated to be about eight years old. The owner's account indicated frequent difficult or painful urination and hematuria during recent weeks, and lately a loss of appetite. A routine qualitative examination of the urine, made by students, showed albumin and red blood-cells. Unfortunately the case received no medical attention during its earlier course.

REFERENCE

[1]MacCallum, W. G.: Text-Book of Pathology. (6th ed., W. B. Saunders, Philadelphia, 1936), p. 433.

OBSERVATIONS ON BANG'S DISEASE*

By W. E. MARTIN, *Wapakoneta, Ohio*

I recently attended a meeting of veterinarians and heard papers and discussions on the subject of Bang's abortion disease. In so many of the articles that I have read, and in talks that I have

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heard on that subject in recent years, the experiences of the authors with Bang's disease have been so much at variance with my own that I am at a loss to explain it.

My first experience with Bang's disease occurred early in the century, on my home farm. At that time, infectious abortion in cattle was discussed and also "cussed" in the farm press. Our farm herd aborted calves for one year, and then the disease died out. I have practiced my profession in the county in which I was born, for over 30 years, and there has not been a year when I was not in contact with Bang's disease, to a greater or lesser extent.

The farm herds in the part of the county over which my practice extends contains from five to 50 head of cattle. All of the common breeds are represented. I coöperate with the federal-state plan of eradication, although I do not believe it is a wise procedure for every herd-owner to follow.

About 15 years ago, Bang's disease broke out in a herd of 35 Holstein cows. I prescribed a program of sanitation which was carried out as one would expect it to be carried out under the conditions existing on the average farm. Abortions occurred in possibly 25 per cent of the cows the first year, and in a much lower percentage the second year. After a few years, the disease died out and has not recurred.

A client had a purebred Jersey herd of 15 cows in which there had never been an abortion. He purchased a bred heifer which aborted. Blood samples were taken from the entire herd and sent to the State Laboratory for the agglutination test. The aborting heifer was the only reactor. I begged him to dispose of her but he refused to do so. She was isolated for three months and then returned to the herd. She has had six healthy calves since the abortion and is still a reactor. There are now three reactors in the herd. The second abortion in this herd occurred last December in a nonreacting heifer.

A purebred Shorthorn herd under my care began to show abortions and was blood-tested. The reacting cattle were moved to another farm. After three years, a reactor is very seldom found in the nonreacting herd, and there are no abortions in the reacting herd. There are no retained placentas. The calves from the reacting herd that test clean at four to six months of age are placed in the clean herd. No vaccines have been used in any of the herds mentioned. I do not suppose I have used 20 doses of abortion vaccine in my life.

In my experience, Bang's abortion disease has been a self-limiting disease, and aside from a possible public health aspect I fail

to see anything in it to get unduly excited about. As I said in the beginning, my experience with Bang's disease has apparently been different from that of other veterinarians. Maybe that accounts for my temerity in making the concluding statement.

COCCIDIOIDAL GRANULOMA IN CALVES*

By C. L. DAVIS *and* GEO. W. STILES

Pathological Division, Bureau of Animal Industry U. S. Department of Agriculture, Denver, Colo.,

and A. N. MCGREGOR

Meat Inspection Division, Bureau of Animal Industry U. S. Department of Agriculture, Los Angeles, Calif.

In a recent article,[1] the authors reported for the first time the occurrence of pulmonary coccidioidal granuloma in the adult bovine. Previous to that, the disease had been observed in cattle ranging in age from one to six years and the lesions were confined to the thoracic lymph-nodes. Since pulmonary lesions were first demonstrated in cattle six years of age, it was assumed that the primary infection occurred at a somewhat earlier age in the thoracic lymph-nodes and that the probability of the disease spreading from lymph-glands to other tissues would increase if affected animals were not slaughtered at an early age. However, a recent finding of Dr. F. C. Kellogg, of the federal Meat Inspection Division, at Los Angeles, California, is noteworthy. He encountered lesions of coccidioidal granuloma with pulmonary involvement in calves eight to ten months of age.

REPORT OF CASES

On January 6, 1938, 48 Hereford calves, eight to ten months old, from Madera County, California, were slaughtered for food purposes at a federally inspected establishment in Los Angeles. Upon postmortem examination conducted by Dr. Kellogg, 23 of the 48 calves exhibited lesions in the thoracic lymph-glands, suspected of being coccidioidal granuloma. Seven of the animals showed slight granulomatous lesions in the lungs in addition to the lymph-gland involvement. Lesions from several cases were submitted to the laboratory for examination and the diagnosis of coccidioidal granuloma was confirmed bacteriologically and by histologic means.

*Received for publication, March 5, 1938.

COMMENTS

The above findings alter somewhat the viewpoint expressed in our recent article in which it was inferred that the probability of finding lesions of wider dissemination would increase if affected animals were permitted to live longer. While such reasoning appears logical, the fact that lesions of coccidioidal granuloma were demonstrated in cattle under one year of age shows that the disease may affect the young bovine and that pulmonary lesions may occur in the early stages of the disease.

REFERENCE

[1]Davis, C. L., Stiles G. W., and McGregor, A. N.: Pulmonary coccidioidal granuloma. A new site of infection in cattle. Jour. A. V. M. A., xci (1937), n. s. 44 (2), pp. 209-215.

RABIES IN TWO GRAY SQUIRRELS*

By H. D. PRITCHETT, *Philadelphia, Pa.*

On May 19, 1937, a young gray squirrel, captured on Andrill Terrace in Charlotte, N. C., was placed in the care of the writer for observation and possible treatment.

History: The history submitted with the animal was negative, as it had been captured only as a result of its inability to escape. No one had knowledge of this rodent having been molested by dogs or other animals and its body bore no traumatic evidence of such an assault, or the results of any other encounter. It was in good physical condition and well developed. It is also unknown whether or not the captor or captors were bitten by the squirrel.

Symptoms: Complete posterior bilateral and caudal motor paralysis were established. The animal moved by dragging the ventral aspect of the body on the ground, propulsion being affected with the fore limbs. Locomotion, however, was performed with ease, and seclusion constantly sought. The squirrel would chatter incessantly when disturbed and was very aggressive, which of course was only natural. The animal was incarcerated in a thick cardboard carton to await developments, during which time food and water were refused. When observed on the following day, it had apparently attempted to escape by gnawing at the loose flaps of the carton; and during the second night, it eventually cut out a hole in the same sufficiently large to permit the protrusion of the head, in which position it was found dead. If mandibular paralysis ever existed, it was not observed.

*Received for publication, March 12, 1938.

Diagnosis: At autopsy it was found that postmortem decomposition had obliterated the identification of, if any, visceral pathological lesions; but there were no pelvic or spinal fractures, or cranial traumatism. Neither was parasitism in evidence macroscopically. The brain was examined on May 21, 1937, by Dr. S. T. Walton, bacteriologist, Health Department of Charlotte, according to the Williams modified technic,[1] and found to be positive for rabies.

Second Case

On January 21, 1938, an individual living on Roslyn Avenue, also in Charlotte, was severely bitten and her garments badly torn by a pet gray squirrel. The brain of this specimen also was examined by Dr. Walton and found to be positive for rabies. An account of this finding and further details appeared in the *Charlotte Observer* of January 22.

Again, on February 27, another article appeared in the same newspaper, stating that a local game warden had shot two squirrels at different points within the city limits of Charlotte, for the purpose of determining their status as to rabies. Both were found to be negative on examination at the above laboratory. Such evidence, though, is of questionable value, inasmuch as these animals were apparently normal before execution, and probably not even in the incubation stages of the disease.

Reference

[1]Williams, A. W.: In: Hallock, W.: Pathogenic Microörganisms, p. 91 (1933).

DIFFICULT SWALLOWING IN A HORSE*

By L. P. Doyle *and* J. F. Bullard
Department of Veterinary Science
Purdue University Agricultural Experiment Station
LaFayette, Indiana

The subject of this report was a horse about five years old, which was said to have been unable to swallow, or swallowed with difficulty, for a period of approximately nine days. Immediately preceding the onset of difficult deglutition, the animal was off feed and showed an elevation of body temperature for one or two days. These symptoms began during the second week of November. No other horse on the farm where this one was or on the neighboring farms was known to be affected.

*Received for publication, March 12, 1938.

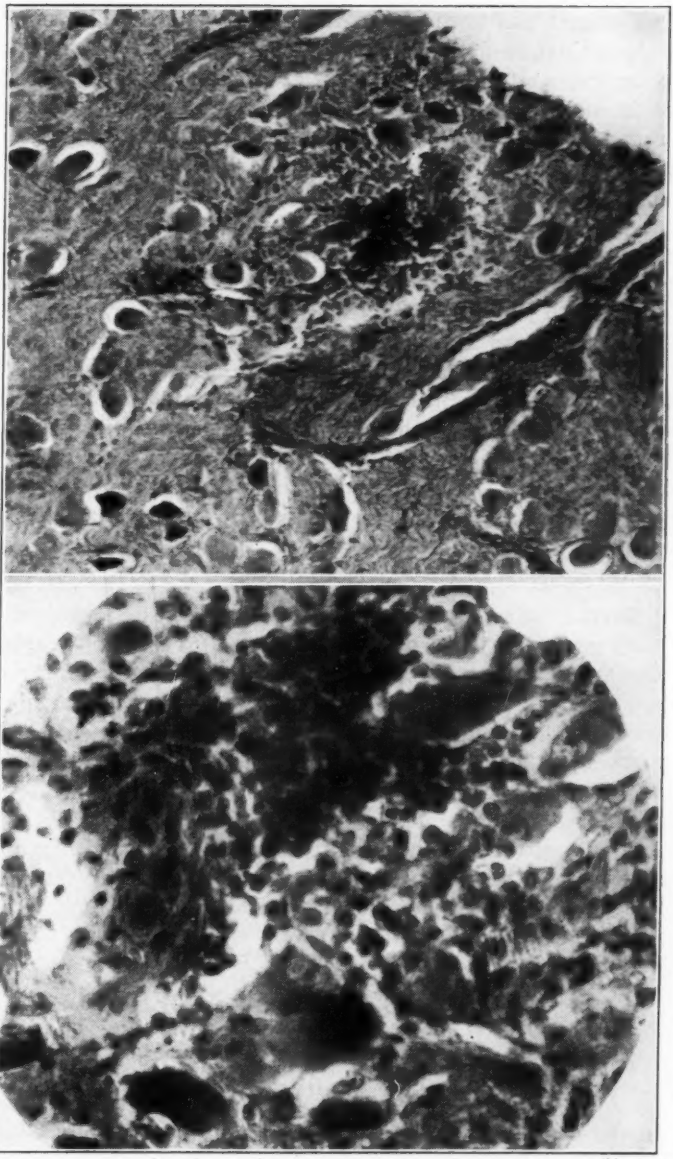

FIG. 1 (above). Photomicrograph of a section of the petrous ganglion, showing an area of round-cell infiltration.

FIG. 2 (below). A higher magnification of a portion of the infiltrated area in the ganglion. It shows some of the invading cells within the ganglion cells, suggesting neurophagic activity.

The animal had been given water and some food by means of the stomach-tube daily for several days. When the horse was seen by the writers, it was not showing any well-defined symptoms in addition to the dysphagia. The owner had become discouraged regarding the prospects for recovery and requested that the animal be killed and examined.

The postmortem examination did not show gross lesions which would account for the difficult deglutition. Microscopic examination was made of the various portions of the brain and of the hypoglossal and glossopharyngeal nerves. No microscopic lesions were found in the brain or in the nerve trunks. However, definite lesions were found in the ganglion of the glossopharyngeal nerve. In the petrous ganglion, there were areas which showed well-marked, round-cell infiltration, together with evident degenerative changes in some of the ganglion cells in the infiltrated areas. In some instances the ganglion cells were largely replaced by infiltrating cells. Numerous other ganglion cells, not in the cell-infiltrated areas, appeared shrunken and were more deeply stained than normal cells.

Discussion

Difficult swallowing or inability to swallow is considered to be a fairly common symptom in the horse. In cases of encephalitis, accompanied by lethargy, a disinclination to swallow may easily be mistaken for inability to swallow or deglutitory paralysis. The microscopic findings in this case are seen to be of a nature to suggest an infective process rather than a toxic condition. This is of interest in view of the tendency to consider deglutitory disturbances as a symptom of botulism. Moreover, the findings in this case suggest that a rather extensive and careful search of the nervous mechanism may be necessary in order to find a satisfactory pathologic explanation for paralytic symptoms.

BRAIN ABSCESS IN A DEER*

By E. M. Joneschild, *Helena, Mont.*
Montana Livestock Sanitary Board Laboratories

In the heavily forested area in the vicinity of Libby, Montana, a blacktail buck deer was observed by the deputy game warden to have impaired senses of sight, smell and hearing and carrying its head to one side.

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Recent newspaper reports of infectious keratitis in deer in Colorado had caused the Montana Fish and Game Commission some concern. The deputy state game warden at Libby immediately got in touch with the State Fish and Game Warden who reported the matter to Dr. W. J. Butler, State Veterinary Surgeon. The writer was detailed from the Montana Livestock Sanitary Board laboratories at Helena to investigate the condition.

The deer was very easily caught, although strictly a wild animal. It was found to have impaired senses of sight, smell and hearing. The head was continually held toward the left side and the animal walked in circles to the left when driven. It was very docile and made no attempt to bolt or get away when left alone. As far as

FIG. 1. Blacktail buck deer with abscess of brain.

could be determined, it was practically blind, although there were no symptoms of keratitis or of any infectious-contagious condition. There was no opacity or any pathological condition observed on macroscopic examination of the eye. The animal being helpless and it being determined that it would undoubtedly die from injury, predatory animals or other causes if left alone, it was decided to destroy it and hold an autopsy.

Autopsy disclosed numerous bruised areas in the muscles due to falls. It was known that, the day previous to the autopsy, it had fallen off a cliff. A postmortem inspection of the internal

thoracic and abdominal organs failed to disclose any pathology. However, six tapeworms (*Moniezia* sp?) were found in the duodenum. Four tapeworm cysts (*Cysticercus tennicollis*) were attached to the peritoneum and a few larvae of the deer bot-fly (*Cephenomyia pratti*) were found in the posterior nares.

Upon examination of the brain a large abscess containing about one ounce of thick, greenish pus was found in the cerebrum. The autopsy demonstrated that the bacterial infection had gained entrance through the antler core, either at the time of shedding or soon thereafter. This is rather an interesting finding, as it is not improbable that it is not an uncommon occurrence for infection to gain entrance into brain tissue through the antler core in animals that shed their horns.

Laboratory examination at the Montana Livestock Sanitary Board laboratories at Helena demonstrated the abscess to be due to the *Corynebacterium pyogenes* with *Staphylococcus albus* and *S. aureus* as secondary invaders.

Big Dog Show in Chicago

The entry list for the International Kennel Club show, which opens at the International Live Stock Amphitheatre on April 2, represents 28 states and three Canadian provinces. Among the 1,169 dogs entered are 94 cocker spaniels, 60 chows, 54 Doberman pinschers, 54 English setters, 52 Boston terriers, 44 English springer spaniels, 44 Scottish terriers, 44 fox terriers and 40 Irish setters. The rest of the entries are divided among 70 breeds.

Remedy for Mange

Dr. T. A. Elliott, state veterinarian of Idaho, made the front page of the Chicago *Tribune* recently. An Associated Press dispatch reported that Dr. Elliott had received a postal card from a resident of the Windy City telling him how to treat the mange-affected elk in Idaho's vast primitive area. The remedy was simple enough—Epsom salt and borax, but the Chicagoan failed to tell Dr. Elliott how to get the remedy and the elk acquainted.

The 64th annual Kentucky Derby will be run at Churchill Downs, Louisville, Ky., Saturday, May 7, 1938.

THE CELLULAR RESPONSE IN ACQUIRED RESISTANCE IN GUINEA PIGS TO AN INFECTION WITH PIG ASCARIS. K. B. Kerr. Amer. Jour. Hyg., xxvii (1938), p. 28.

The average lethal dose of the pig ascaris, *Ascaris lumbricoides suum,* for guinea pigs was between 30 to 50 eggs per gram body weight and the average time after infection that death occurred from verminous pneumonia was eight days. Experiments were conducted to determine whether a true acquired immunity can be established against the pig ascaris. Both single and multiple sublethal doses, given at varying intervals, induced a resistance to an otherwise lethal dose of ascaris eggs. The resistance was sufficient to permit guinea pigs to survive an infection of about twice the average lethal dose but was not so effective against a second test infection. A more marked cellular response was found in the cecal wall of the resistant guinea pigs than in that of the non-resistant. More larvae were destroyed in the liver of resistant guinea pigs by typical foreign-body reactions than in non-resistant animals. Larvae were destroyed in the lungs of non-resistant guinea pigs. A greater degree of hemorrhage in the lungs occurred in non-resistant guinea pigs than in resistant ones. Due to the number of larvae found involved in the cellular response in the liver, it was concluded that this organ was most important in walling off and destroying larvae.

ATTEMPTS TO INDUCE AN ARTIFICIAL IMMUNITY AGAINST THE DOG HOOKWORM, ANCYLOSTOMA CANINUM, AND THE PIG ASCARIS LUMBRICOIDES SUUM. K. B. Kerr. Amer. Jour. Hyg., xxvii (1938), p. 52.

Attempts were made to induce an artificial immunity in mice to the dog hookworm, *Ancylostoma caninum,* and in guinea pigs to the pig ascaris, *Ascaris lumbridcoides suum.* None of the four antigens used in the attempts to immunize against the dog hookworm gave significant results. Ten of the 20 guinea pigs subjected to a series of injections with dried powdered ascaris antigen survived test infections. The author suggests the size of the

test infection and the type of antigens used as being responsible for failure to stimulate an obvious resistance.

STUDIES ON THE PASSIVE TRANSFERENCE OF ACQUIRED RESISTANCE TO THE DOG HOOKWORM AND PIG ASCARIS. K. B. Kerr. Amer. Jour. Hyg., xxvii (1938), p. 60.

Experiments are reported in which attempts were made to transfer passively an acquired resistance against the dog hookworm in mice and dogs, and a similar resistance against the pig ascaris in guinea pigs to other guinea pigs. Three of the six experiments on resistance to hookworm were negative. In the other three, seven of the 17 mice which received "immune" serum survived the test infection, while 17 of the 18 control mice died. In two experiments on resistance to the pig ascaris, three of the seven guinea pigs which received immune serum survived the test infection, while six of the seven controls died. Although the results are not considered conclusive, the author suggests that they add weight to the suggestion that a humoral antibody is present in resistance to a nematode infection which has been acquired from previous infections.

SELF LIMITATION AND RESISTANCE IN TRICHOMONAS FOETUS INFECTION IN CATTLE. Justin Andrews. Amer. Jour. Hyg., xxvii (1938), p. 149.

Experimental evidence indicates spontaneous cure and refractiveness in genital trichomoniasis of cattle and the development of acquired resistance to this infection. The susceptibility of recovered animals is altered so that they are no longer receptive to the infection even when repeatedly introduced. Therapeutic measures such as the irrigation of the infected genital tract with disinfectant solutions must have their efficiency tested and noted against self-cure. The possibility of control in heifers by deliberate infection before sexual maturity is being investigated. Cattle-breeders in some instances report increased breeding efficiency of cows that have been infected over heifers. It is suggested that endemic trichomonad infection in a herd may have a real bearing on the problem of delayed conception as well as other reproductive defects of susceptible cattle.

MEAL BEETLES AS INTERMEDIATE HOSTS OF POULTRY TAPEWORMS. Margery W. Horsfall. Poultry Sci., xvii (1938), p. 8.

The meal beetles, *Tribolium castaneum* and *Tribolium confusum*, were experimentally infected with three species of com-

mon poultry cestodes, *Hymenolepsis carioca, Choanotaenia infundibulum* and *Raillietina cesticillus*. The resulting cysticercoids were fed to laboratory-raised chickens and the adult cestodes recovered upon postmortem examination. These two species of beetles which are widely distributed usually feed upon cereal products and were found to eat gravid segments of *Raillietina cesticillus* even when abundant meal was available. Infected meal beetles may be shipped long distances and birds may be infected at any season of the year by eating the parasitized beetles.

A Study of the Bacteria Occurring in Commercial Fowl-Pox Vaccine. Herman A. Dettwiler and Floyd S. Markham. Poultry Sci., xvii (1938), p. 46.

Twenty samples of fowl-pox vaccine, representing the stock of seven widely separated manufacturers, were examined bacteriologically, the bacteria count per gram of material ranging from 23,000,000 to 1,200,000,000. No commonly recognized poultry pathogen was found. The conclusion is reached that the bacteria found in commercial fowl-pox vaccine are of the "opportunist" type and that post-vaccination mortality in flocks should not be attributed to pathogenic bacteria present in such products.

Susceptibility and Resistance of Some Strains of Chickens to Fowl Leukosis. H. L. Wilcke, C. D. Lee and Chas. Murray. Poultry Sci., xvii (1938), p. 58.

The causative agent of fowl leukosis is extremely virulent, as evidenced by the fact that progeny of seven out of eight matings were equally susceptible to the disease whether injected with the virus or simply exposed to the disease by contact. The progeny of individual hens are quite variable in their resistance or susceptibility to fowl leukosis. It is possible to develop by selection strains of birds resistant or susceptible to fowl leukosis.

Experiments in the Trasmission of Fowl Lymphomatosis by Inoculation. J. R. Beach. Poultry Sci., xvii (1938), p. 67.

The author failed to demonstrate the transmissibility of avian lymphomatosis in two concurrent series of transmission trials with two strains of the disease. Birds were inoculated with neuro-lymphomatosis and visceral lymphomatosis material. Neural and visceral lymphomatosis lesions developed in two groups in the ratio of 2 to 1 and 3 to 1, respectively. Control birds developed neural and visceral lesions as well but in a smaller

percentage of cases. The results do not signify the transmissibility of lymphomatosis. The author suggests that the experiments indicate the necessity in lymphomatosis in transmission experiments of (1) the use of pedigreed chicks; (2) predetermining the susceptibility or resistance of chicks used for such experiments and the hatching and rearing of chicks apart from others and under conditions which preclude natural exposure to the disease.

CHEMICAL INVESTIGATIONS ON THE ACTIVE PRINCIPLES OF THE PHENOMENON OF LOCAL SKIN REACTIVITY TO BACTERIAL FILTRATES. II. Physicochemical properties. Gregory Shwartzman and S. A. Morell. Jour. Exp. Med., lxvii (1938), p. 1.

Physicochemical properties of the active principles of the phenomenon of local skin reactivity to bacterial filtrates were studied. Ultrafiltration through Zsigmondy filters of graded porosites showed that the active substances were retained by membranes finer than 100 to 120 seconds. The average porosity of this filtration endpoint represents a particle size of about 50 to 100 millimicrons. The active principle was fractionally precipitated by ammonium sulfate. In cataphoresis experiments the substances suspended in culture filtrates migrated to the anode at pH 3.0 or below, while at pH 4.0 or above reversal to the cathode occurred.

STUDIES ON INFLAMMATION. XIV. Isolation of the factor concerned with increased capillary permeability in injury. Valy Menkin. Jour. Exp. Med., lxvii (1938), p. 129.

A factor is present in inflammatory exudates which induces an increase in the permeability of normal capillaries. Its liberation and presence in exudates offers a reasonable explanation for the mechanism of increased permeability of small vessels in injured tissue. This factor in purified form is a crystalline, doubly refractive, nitrogenous substance and evidently is not a protein. The active material is dialyzable and precipitated by concentrated ammonium sulfate. Evidence indicates that it is an intermediary breakdown product of protein metabolism probably belonging to the group of relatively simple polypeptides.

STUDIES ON INFLAMMATION. XV. Concerning the mechanism of cell migration. Valy Menkin. Jour. Exp. Med., lxvii (1938), p. 145.

A crystalline nitrogenous substance recovered from inflammatory exudates induces in cutaneous tissue a rapid migration of

polymorphonuclear cells through the endothelial wall of small vessels. The tentative name of leukotaxine is proposed. The substance stimulates the basic sequences of inflammatory reaction—increased capillary permeability followed by the outward migration of polymorphonuclear leukocytes. Comparison of the effect of various inflammatory irritants (turpentine or aleuronat) with that of leukotaxine indicates that the migration of cells does not seem to be primarily referable to the initial increase in vascular permeation. Leukotaxine is definitely chemotactic. It induces an active migration of leukocytes into capillary glass tubes containing the substance.

STUDIES ON INFLAMMATION. XVI. On the formation of a chemotactic substance by enzymatic action. Valy Menkin. Jour. Exp. Med., lxvii (1938), p. 153.

Blood-serum digested by tryspin yields split products which when injected into the skin of normal rabbits increase the permeability of small cutaneous vessels. This is likewise followed in the affected tissue by prompt local migration of polymorphonuclear leukocytes. The tryptic digest of blood-serum can be purified to yield a crystalline substance which alters the permeability of the capillary wall and favors local migration of leukocytes in a manner similar to leukotaxine. Leukotaxine or a substance having identical biological properties appears to form as the result of the proteolytic enzymatic action on an otherwise relatively inactive blood-serum.

STUDIES ON THE RELATIONSHIP OF THE SEX HORMONES TO INFECTION. I. The effect of the estrogenic and gonadotropic hormones on vaccinia and the spreading factor. Douglas H. Sprunt, Sara McDearman and James Raper. Jour. Exp. Med., lxvii (1938), p. 159.

The estrogenic hormone after being given for one week slows up the spread of India ink on the surface of the skin but allows it to reach and exceed a normal spread after 24 hours. After the injection of the estrogenic hormone for three weeks, the spread of India ink was much less than in control animals. The resistance of the rabbit to vaccinia is increased if the rabbit was castrated and then given the estrogenic hormone for a period of three weeks before being vaccinated. The experiments on the action of gonadotropic hormone on the spread of India ink did not agree.

CONCENTRATION AND PURIFICATION OF BACTERIOPHAGE. John H. Northrop. Jour. Gen. Physiol., xxi (1938), p. 335.

The author isolated a nucleoprotein, which is closely associated with bacteriophage activity, from lysed staphylococci. The protein is very unstable and is denatured by acidity greater than pH 5.0 and by temperatures over 50 degrees C. for five minutes. It is digested by chymo-trypsin but not by trypsin. The rate of diffusion and sedimentation of the protein is the same as that of bacteriophage. The loss in activity when susceptible living or dead bacteria are added to a solution of the protein is proportional to the loss of protein from the solution. Non-susceptible bacteria remove neither protein nor activity. It is suggested that the formation of bacteriophage may be more simply explained by analogy with the autocatalytic formation of pepsin and trypsin than by analogy with the far more complicated system of living organisms.

Chappel Award

Mrs. George Bethune Adams, age 88, has been awarded the 1937 Chappel Award for distinguished service to dogdom. The award took place at the Ellin Prince Speyer Hospital, in New York City. For more than a score of years Mrs. Adams served as Manager and Resident Director of the Speyer Hospital.

In placing her name before the judges, a committee from the Dog Writers Association wrote about Mrs. Adams, "her unremitting work of day and night service to the relief of all animals, especially dogs; her courage and high sense of devotion to duty, coupled with her continued and prolonged efforts toward a better understanding between dogs and their owners and the example she has set for others who work so that man's duty to all domestic animals may in some better degree be met."

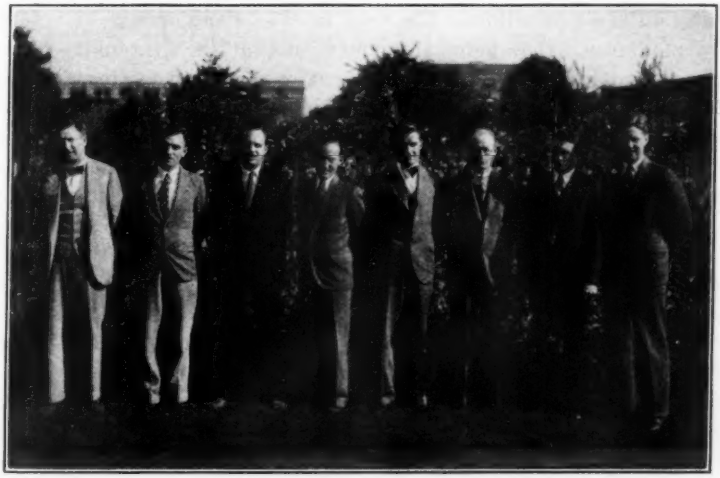
The judges whose vote selected Mrs. Adams were as follows: Mrs. M. Hartley Dodge, Madison, N. J.; Mr. George F. Foley, Philadelphia, Pa.; Mr. Roland Kilbon, Brooklyn, N. Y.; Mr. C. E. Harbison, New York; Capt. Will Judy, Chicago, and Mrs. Josephine Z. Rine, Caldwell, N. J. Previous winners of the annual Chappel Awards are: Mr. George F. Foley, Mr. James W. Spring and Mrs. M. Hartley Dodge.

Only the brave know how to forgive. A coward never forgave; it is not in his nature. —*Laurence Sterne*

Doctor Simms to Alabama

Dr. B. T. Simms has resigned as Professor of Veterinary Medicine and Head of the Veterinary Department at Oregon State College, Corvallis, to accept the position of Director of the Regional Animal Disease Research Laboratory, which is being established by the U. S. Bureau of Animal Industry at Auburn, Ala. Dr. Simms went to Oregon in 1913 and developed the Veterinary Department to the point where it was the third largest of its kind in the United States in those institutions not offering a course in veterinary medicine. It is larger than either the Animal Husbandry or the Dairy Husbandry Department at Corvallis, probably the only place in the country where this is true.

At the meeting of the Willamette Valley Veterinary Medical Association, held at the Multnomah Hotel, in Portland, on March 9, the veterinarians of Oregon presented Dr. Simms with a watch as a token of their appreciation of his 25 years of serv-



THE CORVALLIS VETERINARY OCTETTE

ice to the live stock interests of the state. Dr. Simms is scheduled to report for his new duties at Auburn on April 1.

The accompanying photograph is reproduced from the JOURNAL of December, 1930, and shows the eight veterinarians who were members of the staff of the Veterinary Department at Corvallis at about that time. From left to right they are: Drs. J. N. Shaw (Wash. '17), Fred C. Myers (Colo. '24), A. M. McCapes (Colo. '27), F. M. Bolin (Iowa '29), O. H. Muth (Mich. '29), W. T. Johnson (Wash. '17), B. T. Simms (A. P. I. '11) and E. M. Dickinson (O. S. U. '20).

Drs. Shaw, Myers, Bolin and Muth are still at Corvallis, Dr. McCapes is now in Missouri, Dr. Johnson died a short time ago, and Dr. Dickinson is in California.

Ohio State Veterinary Conference

The eleventh annual conference of the College of Veterinary Medicine, Ohio State University, will be held June 15, 16 and 17, 1938. This year, as formerly, the program is quite an extensive one and includes some of the foremost veterinarians and educators in this country. The entire three-day period will be devoted to the discussion of topics of vital importance to every veterinarian who can be present on this occasion. A special effort has been made in arranging the details of the program so that its quality and variety will appeal to all veterinarians.

The program this year has been formulated for the purpose of giving to the practitioner the very latest developments in veterinary medicine. It is generally conceded that such intensive short courses provide the necessary educational opportunities for the busy practitioner. The College of Veterinary Medicine hopes it will be possible for a large number to avail themselves of this opportunity to receive the very latest information concerning many of the real problems in their various fields of activity.

These short courses are sponsored by the College of Veterinary Medicine, believing it can render a real service to the live stock industry through the veterinary profession. In the past years, the attendance has been very gratifying to those who have been interested in promulgating these courses. The interest manifested in the programs and the various discussions entered into by those in attendance have been a source of much gratification also. An invitation is hereby extended to all veterinarians to be present on this occasion to receive, we feel sure, much of value from an educational point of view.

Regular Army

Captain Geo. T. Price is relieved from duty at Fort Sam Houston, Texas, effective at such time as will enable him to proceed to Charleston, S. C., and sail on transport scheduled to leave that port on or about June 23, 1938, for the Panama Canal Department, for duty with the Veterinary Corps.

Lt. Colonel Frank B. Steinkolk is assigned to duty at the general dispensary, U. S. Army, Washington, D. C., and to additional duty as attending veterinarian, Army War College; Bolling Field, D. C., and Fort Washington, Md., effective upon completion of his present tour of foreign service in Panama.

Captain Stanley M. Nevin is assigned to duty at Barksdale Field, La., effective upon completion of his present tour of foreign service in the Philippine Department.

Captain Albert A. Roby, Jr., is relieved from duty at Fort Bliss, Texas, is then assigned to station at Fort Hoyle, Md., and will proceed at the proper time to San Francisco, Calif., and sail on the transport scheduled to leave that port on or about July 30, 1938, for New York, N. Y.; upon arrival in New York will proceed to Washington, D. C., and report to the commanding general, Army Medical Center, for temporary duty for a period of approximately five months for the purpose of pursuing a course of instruction at the Army Veterinary School. Upon completion of this temporary duty, Captain Roby will proceed to Fort Hoyle, Md., and report for duty.

1st Lt. Thomas C. Jones is relieved from further assignment and duty at the Presidio of Monterey, Calif., is then assigned to the Army Veterinary School, Army Medical Center, Washington, D. C., and will proceed at the proper time to San Francisco, Calif., and sail on transport scheduled to leave that port on or about July 30, 1938, for New York, N. Y.; upon arrival in New York will proceed to Washington, D. C., and report to the commanding general, Army Medical Center, for duty for the purpose of pursuing a course of instruction at the Army Veterinary School.

1st Lt. Donald C. Kelley is relieved from duty at Fort Riley, Kans., effective in time to comply with this order, is then assigned to the Army Veterinary School, Army Medical Center, Washington, D. C., and will proceed at the proper time to Washington, D. C., and report to the commanding general, Army Medical Center, on September 15, 1938, for duty for the purpose of pursuing a course of instruction at the Army Veterinary School.

Lt. Colonel Chauncey E. Cook is relieved from further assignment and duty at the general dispensary, United States Army, Washington, D. C., and from additional duty as attending veterinarian at Fort Humphreys, D. C., Fort Washington, Md., and Bolling Field, D. C., effective on June 19, 1938, and assigned to Fort Benning, Ga., for duty.

Lt. Colonel Seth C. Dildine is relieved from further assignment and duty in China and the Philippine Department, effective at such time

as will enable him to comply with this order, and is then assigned to duty at Fort Lewis, Wash. He will proceed to the United States on the U. S. A. T. *Meigs*, and upon arrival in the United States, will proceed to Fort Lewis, Wash., and report for duty.

Major Herbert K. Moore is relieved from duty at the New York Port of Embarkation, Brooklyn, N. Y., effective in time to comply with this order, is assigned to the Quartermaster Depot, Seattle, Washington, and will sail on transport scheduled to leave New York, N. Y., on or about March 25, 1938, for San Francisco, California; upon arrival in San Francisco will proceed to Seattle, Washington, and report to the commanding officer, Quartermaster Depot, for duty.

Lt. Colonel Frank C. Hershberger is relieved from duty at Fort Hamilton, N. Y., effective on or about March 15, 1938, is then assigned to duty at the New York Port of Embarkation, Brooklyn, N. Y., and additional duty as attending veterinarian, Fort Hamilton, N. Y.

Veterinary Reserve Corps

NEW ACCEPTANCES

Martin, Ellis James.....1st Lt....Millerville, Ala.
Thome, Marvin Thorwald, Sr. 1st Lt....408 State Office Bldg., Columbia S. C.

PROMOTIONS

To

Butz, Harrison Wm. Jr.....1st Lt....3764 Vine St., Cincinnati, Ohio.
Holland, Ed Richard.....1st Lt....P. O. Box 1086, Sacramento, Calif.
Mahaffy, James Roberts.....Lt. Col....P. O. Box 2729, Honolulu, T. H.

NEW ASSIGNMENTS TO ACTIVE DUTY WITH CCC

Wiseman, Edwin Stranel.....1st Lt....Hq. Southern Dist. CCC, Richmond, Va.
Pundt, Werner .....1st Lt....Pres. San Francisco, Calif.
Taylor, Albert Andrew.....1st Lt....Pres. San Francisco, Calif.

TERMINATION OF ASSIGNMENT TO ACTIVE DUTY WITH CCC

Omohundro, Richard Eugene..1st Lt....Hq. Southern Dist. CCC, Richmond, Va.
Evans, Tom .....Capt....New Mexico Dist. CCC, Fort Bliss, Tex.
Andrews, Asa R.....Capt....Pres. San Francisco, Calif.
Hartzell, Harold P.....1st Lt....Fort Douglas, Utah.
Saunders, Charles M.....1st Lt....Fort Missoula, Mont.

Make it thy business to know thyself, which is the most difficult lesson in the world.

—Cervantes

The wise course is to profit from the mistakes of others.

—Terence

VETERINARY MEDICAL ASSOCIATION OF NEW YORK CITY

The regular monthly meeting of the Veterinary Medical Association of New York City was held at the Hotel New Yorker, on Wednesday evening, January 5, 1938.

The guest speaker of the evening was Dr. Charles R. Schroeder, in charge of the hospital and laboratory of the New York Zoölogical Park, who explained the veterinary activities of such an institution. New specimens, whether purchases or gifts, must go through quarantine and isolation periods in accordance with regulations of the U. S. Bureau of Animal Industry. Final housing must be given a great deal of attention due to changes in life zones. Imported animals are kept under conditions of proper temperature and humidity as far as possible. Disinfection of the Zoo consists primarily in physical cleanliness. It has been found possible to control odors to a large extent by the use of an essential oil.

Dr. Schroeder stressed the proper handling of food and stated that when cases of food poisoning occur among the animals, they are usually caused by organisms of the genera *Staphylococcus, Salmonella* and *Proteus*. Vitamin feeding is also practiced. Fish-liver oil is purchased on a basis of units of vitamins A and D, rather than on a basis of cost per pound or gallon. The high content of vitamin D is essential throughout the year for housed specimens.

Food containers are cleaned and sterilized in a hot solution of 2 per cent lye. Subsequently, four rinsings are necessary to insure the freedom of the containers from any of the lye solution. Various parasites are controlled by the use of santonin, tetrachlorethylene, chenopodium and thymol.

Dr. Schroeder also discussed the use of antiseptics, especially nembutal, in connection with surgical operations at the Zoo. He recommended shredded sugar cane for bedding. This is a sterile product, non-dusty and absorbent.

Still pictures were shown of the hospital, laboratory and kitchen at the Zoo. These were followed by motion-pictures of two male mountain gorillas which were in captivity in San Diego, Calif.

This unusual and interesting presentation was greatly appreciated by the members, and a rising vote of thanks was extended to Dr. Schroeder. JOSEPH B. ENGLE, *Secretary.*

INTERMOUNTAIN LIVE STOCK SANITARY ASSOCIATION

The eleventh annual meeting of the Intermountain Live Stock Sanitary Association was held in Ogden, Utah, January 6-8, 1938, with Dr. Oscar Wennergren, of Logan, presiding. Hon. Harmon W. Perry, Mayor of Ogden, appropriately welcomed the assembled veterinarians to the city. Dr. J. W. Chambers responded to the address of welcome. In his presidential address, Dr. Wennergren reviewed the accomplishments made in veterinary science and mentioned some of the many prospects of the future.

Dr. Frank Thorp, of Colorado State College, presented further details on oat hay poisoning. Since most cattle that succumb to the disease show a positive reaction to hydrocyanic acid in the rumen, by the picric acid test, poisoning by hydrocyanic acid is under suspicion, although other studies have led the investigators to conclude that the disease is not one of typical HCN poisoning, as usually observed.

Prof. A. J. Morris, of the Utah State Agricultural College, gave an interesting discussion of the factors involved in the production of a high-grade milk. The healthy cow was the first essential listed. Another factor which contributes more than any other toward the successful production of high-quality milk is cleanliness of the milk utensils, according to Prof. Morris.

"Artificial Insemination of Ewes with Transported Semen" was discussed by Dr. E. M. Gildow, of the University of Idaho. Semen shipped 700 miles under refrigeration was found viable and capable of fertilization. Lincoln ewes were found to have longer estrual periods than Southdowns or Hampshires.

Another plant has just been added to the list of plants poisonous to live stock, according to some work done at the Colorado Experiment Station. The plant responsible is *Suckleya suckleyana,* which grows about old water-holes. Death is produced by hydrocyanic acid.

During the evening of the first day, informal discussions were held by several groups, including research workers and those engaged in state and federal control work.

On the second day, Dr. Jean Flint, of Salt Lake City, discussed enteritis in dogs and stressed the importance of always definitely ruling out the possibility of distemper. If this point cannot be decided definitely, it is well to administer anti-canine distemper serum in order to be safe. Dr. Flint found gastro-intestinal lavage helpful in treating cases of enteritis.

Dr. Carl F. Schlotthauer, of the Mayo Foundation, Rochester, Minn., presented some of the newer developments which he had found helpful in connection with surgery in animals. He described the method of administering ether through a tube passed down the trachea. The importance of public health measures and the obligation of the veterinarian in public work were stressed by Dr. Oscar V. Brumley, president of the American Veterinary Medical Association.

On the final day of the meeting, equine encephalomyelitis in the Central West was ably discussed by Dr. H. J. Shore, of Fort Dodge, Iowa. His talk was supplemented by reports made by Drs. R. C. Swalberg, N. C. Spalding and O. L. Montgomery. Utah veterinarians reported unsatisfactory results following the use of serum. Dr. Schlotthauer reported what appeared to be good results following the prophylactic use of biologics prepared from streptococci recovered from horses affected with the disease.

Dr. Don R. Coburn, of the Bear River Migratory Bird Refuge, Brigham, Utah, gave an excellent illustrated discussion of diseases and parasitisms affecting the animals and birds of our public domains and game preserve areas. Dr. Brumley, in his second talk, discussed problems he had encountered in connection with the municipal inspection of foods in Columbus, Ohio. Dr. Schlotthauer gave a report on experiences encountered with certain types of mastitis.

A highly successful banquet featured the evening of January 8, at which Dr. Brumley was the chief speaker.

Officers for the coming year were elected as follows: President, Dr. A. H. Francis, Denver, Colo.; first vice-president, Dr. W. H. Hendricks, Salt Lake City, Utah; second vice-president, Dr. S. E. Nelson, Preston, Idaho; third vice-president, Dr. A. K. Kuttler, Boise, Idaho, and secretary-treasurer, Dr. D. E. Madsen (reëlected), Logan, Utah.

It was decided to hold the 1939 meeting in Salt Lake City.

D. E. MADSEN, *Secretary-Treasurer.*

VETERINARY MEDICAL ASSOCIATION OF NEW JERSEY

The fifty-fourth annual meeting of the Veterinary Medical Association of New Jersey was held at the Hotel Hildebrecht, Trenton, January 12-13, 1938.

A business meeting was held at the beginning of the first session, which was followed by a report of the delegate to the American Veterinary Medical Association House of Representatives, Dr. R. A. Hendershott. Dr. Ellis P. Leonard, of Summit, then read a paper on "Feline Therapeutics and Hospitalization" and Dr. R. E. Shope, of the Rockefeller Institute, Princeton, spoke on "Swine Influenza."

In the afternoon, a small-animal clinic was held under the direction of Dr. John T. McGrann, of Trenton. Demonstrations and operations were performed by Dr. A. R. Theobald, of Cincinnati, Ohio; Dr. Ellis P. Leonard, of Summit, Dr. W. R. Ecker, of Newark, and Dr. J. A. S. Millar, of Deal.

On the evening of January 12, the annual Association Dinner was held, with about 125 present and with many ladies included. Dr. C. J. McAnulty, of Atlantic City, presided. The dinner was the occasion of a tribute to Dr. Wilfred Follansbee Harrison (Amer. '87), of Stockton, who completed, last year, 50 years of uninterrupted practice of and service to veterinary science. Most of these years had been spent in New Jersey, where Dr. Harrison has served the State Association and the profession in many capacities. He has served as a member of the State Board of Veterinary Medical Examiners for many years, as president of the State Association, and as a sort of "perennial" member of the Executive Board of the State Association, where his counsel and judgment have been of great value. President McAnulty, in fitting remarks, presented to Dr. Harrison, on behalf of the Association, an engrossed scroll setting forth his outstanding service to the Association and the live stock interests of the state. A large bouquet of roses was also presented to Mrs. Harrison as a tribute to her splendid participation in the "partnership."

President McAnulty then called upon Mr. Willard H. Allen, the newly appointed secretary of the State Board of Agriculture, who made a brief address, pledging his coöperation in problems of mutual interest. Mr. Charles G. Hopton, of New York City, noted breeder and judge of dogs, was then introduced and was the principal speaker. Following the dinner, an orchestra furnished an opportunity for dancing for the balance of the evening.

On January 13, the following program was presented:

"Bovine Mastitis—Practical Considerations of Diagnosis and Control," by Dr. George H. Hopson, Kings County Medical Milk Commission, Brooklyn, N. Y.

"Poultry Diseases," by Dr. F. R. Beaudette, College of Agriculture, New Brunswick.

"Random Remarks Relating to Selected Conditions in Small Animals and Their Treatment," by Dr. A. R. Theobald, Cincinnati, Ohio.

"Horses and Horse Shows," by Dr. Cassius Way, New York City.

"Coöperative Animal Disease Control in New Jersey," by Dr. R. A. Hendershott, Trenton.

Brief practical discussions and demonstrations were also given as follows:

"Trichomoniasis," Drs. Clarence B. Johnston, Trenton, and J. B. Hagenbuch, Lawrenceville.

"Teat Surgery," Dr. G. H. Kimnach, Hightstown.

"Torsion of Uterus," Dr. J. M. Herron, Bordentown.

All officers of the Association were reëlected as follows: President, Dr. C. J. McAnulty, Atlantic City; first vice-president, Dr. G. H. Kimnach, Hightstown; second vice-president, Dr. P. F. Runyon, Freehold; treasurer, Dr. J. B. Engle, Summit, and secretary, Dr. J. G. Hardenbergh, Plainsboro.

Four new members were elected and the applications of five others were received. Dr. R. A. Hendershott was named as delegate to the American Veterinary Medical Association House of Representatives to serve for two years and the Secretary was named as alternate.

On account of the American Veterinary Medical Association's Diamond Jubilee meeting in New York City, July 5-9, 1938, it was voted to dispense with our usual summer meeting and confine it to a brief business session some time during the A. V. M. A. meeting.

J. G. HARDENBERGH, *Secretary.*

STATE VETERINARY MEDICAL ASSOCIATION OF TEXAS

The twenty-eighth annual meeting of the State Veterinary Medical Association of Texas was held at the Rice Hotel, Houston, January 13-14, 1938, with approximately 200 veterinarians registered. A diversified program presented by local and visiting veterinarians was enjoyed. Physicians took an active part in the program. Dr. George W. Cox, State Health Officer, presented a paper on "The Veterinarian and His Relation to Public Health." Dr. Royall M. Calder, a practicing physician, discussed "Brucel-

losis in Man," and based his talk on incidence, symptoms and treatment of the disease. Dr. Dudley Jackson, who is a graduate in both human and veterinary medicine, discussed "The Relationship of Cancer in Domestic Animals."

The visiting veterinarians included Dr. H. D. Bergman, president-elect of the American Veterinary Medical Association, who brought greetings from the national organization and also discussed "Some Newer Developments in Physiology of Therapeutic Significance." Dr. D. M. Campbell, of Chicago, discussed "The Effect of Nutrition on Unsoundness in Animals." During this discussion he also showed some very excellent lantern-slides prepared by Col. C. W. Greenlee, V. C., U. S. A., to illustrate his talk. Dr. J. W. Patton, of East Lansing, Mich., discussed "Canine Nutrition and Dog Foods" in an illustrated talk. Dr. Ashe Lockhart, of Kansas City, Mo., discussed "The Control of Bang's Disease by Calfhood Vaccination." Dr. B. M. Lyon, of Pearl River, N. Y., presented a paper on "Equine Encephalomyelitis and Its Control." He also showed a film prepared by Dr. Wyckoff, of the Rockefeller Institute, which was greatly enjoyed.

The following papers constituted the remainder of the program:

"Control of Bovine Brucellosis by Milk Inspection," by Dr. Fred P. Jaggi, Texas A. and M. College.

Report of U. S. Live Stock Sanitary Association meeting, by Dr. T. O. Booth, State Veterinarian.

"Heart-worm Infestation in Dogs," by Dr. M. N. Bader, Galveston.

"Skin Diseases of the Feline," by Dr. H. V. Cardona, Fort Worth.

"Some Newer Developments in Small-Animal Practice," by Dr. J. G. Horning, Houston.

"Internal Parasites of Small Animals and Their Treatment," by Dr. H. L. Van Volkenberg, Texas A. and M. College.

During the business meeting the following officers were elected: Honorary President, Dean R. P. Marsteller, College Station; president, Dr. Leon G. Cloud, Fort Worth; first vice-president, Dr. Sam E. Bunton, Del Rio; second vice-president, Dr. W. F. Hayes, Farmersville; secretary-treasurer, Dr. Dee Pearce, Leonard, and corresponding secretary, Dr. M. B. Starnes, Dallas.

Resolutions were adopted approving the proposed changes in the organization of the American Veterinary Medical Association and endorsing resolution 5 adopted by the A. V. M. A. at the Omaha meeting.

Another resolution provided for inviting the American Veterinary Medical Association to hold the 1939 meeting in Dallas. An invitation to hold the semi-annual meeting of the State Veterinary Medical Association of Texas at the Texas A. and M. Col-

lege, extended by Dean Marsteller, was accepted and Dallas was selected for the annual meeting place in 1939.

M. B. STARNES, *Corresponding Secretary.*

KANSAS VETERINARY MEDICAL ASSOCIATION

The thirty-fourth annual meeting of the Kansas Veterinary Medical Association was held in Topeka, January 19-20, 1938, with headquarters at the Jayhawk Hotel. The weather being exceedingly mild, a large percentage of the practitioners of the state were in attendance. There were over 125 veterinarians registered and more than 50 ladies. The meeting was called to order by the President, L. A. Hammers, of Clearwater. In his presidential address, Dr. Hammers held forth great hope for the future of the profession in the state and strongly urged a larger membership in the State Association. He expressed the opinion that if veterinarians would meet and discuss their problems, more often a closer coöperation of all members of the profession would be the inevitable result.

Dr. Chas. W. Bower, of Topeka, gave a joint report of the 1937 meeting of the American Veterinary Medical Association House of Representatives and a progress report from the A. V. M. A. Executive Board. He outlined the business transacted at Omaha and informed the membership concerning the new plan of financing the A. V. M. A. annual conventions. He likewise told of a plan sponsored by the Executive Board to add additional help to the Secretary's office, whereby better coöperation could be had between the A. V. M. A. and practicing veterinarians. Dr. Bower spoke of the plans to revamp the JOURNAL of the A. V. M. A. with a view to making it a more workable instrument for the general practitioner.

Dr. F. B. Jones, of the U. S. Bureau of Animal Industry, stationed in Topeka, spoke concerning the testing of replacement cattle in Bang's disease-free herds.

Equine encephalomyelitis was discussed very thoroughly at the afternoon session of the first day. The pathology of the disease was presented by Dr. C. H. Kitselman, of Kansas State College. In his discussion he brought out the fact that birds, particularly turkeys, are carriers of the virus of equine encephalomyelitis. He also stated that the virus is frequently excreted through the saliva of horses affected with the disease. It would seem that this is a very important point, especially where animals drink from common watering troughs and ponds.

The clinical phases of equine encephalomyelitis, including symptoms and treatment, were discussed by Dr. C. D. Eberts, of Kansas State College. He advised keeping animals in a standing position as long as possible. In order to do this, he described several forms of improvised slings.

Dr. M. L. Dietrich, of Newton, reported that he had used hog wire padded with sacks of straw as a sling. Dr. S. L. Stewart, of Olathe, was of the opinion that, together with serum and vaccine, the control of flies, mosquitoes and other blood-sucking insects is of the utmost importance. Dr. Stewart stated further that biological products are still in the experimental stage and a great deal of work will have to be done before we know just how much good is obtained through the use of biologics.

Dr. A. T. Kinsley, of Kansas City, Mo., led a discussion on swine diseases. He mentioned pseudo-rabies and stated that this disease is more prevalent in the United States than is generally suspected by the average practitioner.

At 7:00 o'clock the evening of the first day, a banquet was held in the Florentine Room of the Jayhawk Hotel. Dr. R. F. Coffey, of Topeka, chairman of the Committee on Local Arrangements, provided a wide variety of entertainment. Mr. Will G. West, Live Stock Sanitary Commissioner of Kansas, addressed the gathering on "Past and Present Coöperation with the Live Stock Sanitary Commissioner's Office and the Veterinarian." Dean R. R. Dykstra gave a very interesting talk on "The Trend of Veterinary Education."

The Very Reverend John W. Day, pastor of Grace Cathedral, spoke on the subject, "What's Doing in Russia." The speaker had spent some time in Russia and he related personal experiences, which held the attention of the entire audience for over an hour. Dr. Kinsley closed the after-dinner speaking with a few timely remarks. Dr. W. A. Adams, of Glasco, acted as toastmaster, which duty he performed quite masterfully.

Thursday morning, Dr. E. J. Frick, of Kansas State College, discussed "Cat Practice." He mentioned the different breeds of cats and also their temperaments. He described a few of the common diseases of cats that the average practitioner must meet sooner or later, and explained his method of handling each one.

"Non-Infectious Skin Diseases of the Canine" was the topic discussed by Dr. H. G. Stephenson, of Independence. He mentioned having seen considerable nervous dermatitis, for which he was accustomed to prescribing phenobarbital as a sedative. He outlined the various types of skin diseases that are common in this part of the country and differentiated them.

Dr. C. C. Morrill, of Kansas State College, presented a paper entitled, "A Practical Test to Detect the Presence of Cyanogenic Glucosides in Vegetable Tissue." This was a very timely presentation, particularly since so many diseases result from prussic acid poisoning, every summer and fall in this state.

"The Newer Conception in Our Knowledge of Virus Diseases" was presented by Dr. Norman J. Pyle, of Pearl River, N. Y. The speaker was of the opinion that much will be learned concerning the behavior of viruses within the next two or three years, as a great deal of research work is being done on virus diseases at the present time.

At the business session, Resolution 5, adopted by the American Veterinary Medical Association, at Omaha, was approved in principle. The 1939 meeting will be held in Manhattan, with Kansas State College as the host.

The new officers are: President, Dr. Kent R. Dudley, of Iola; vice-president, Dr. J. A. Bogue, of Wichita, and secretary-treasurer, Dr. Charles W. Bower (reëlected), of Topeka.

CHARLES W. BOWER, *Secretary.*

MISSISSIPPI STATE VETERINARY MEDICAL ASSOCIATION

The thirty-second annual meeting of the Mississippi State Veterinary Medical Association was held in Jackson, January 24-25, 1938. The meeting was called to order by President Van F. Bess, of Vicksburg, and the invocation was given by Dr. I. S. McAdory, of Auburn, Ala. Mayor Walter A. Scott welcomed the Association to Jackson and gave the members the keys to the city. Dr. E. S. Brashier, State Veterinarian, responded to the address of welcome.

Dr. S. L. Stewart, of Olathe, Kan., opened the program with his paper, "Bang's Disease from the Practitioner's Standpoint." Dr. Stewart is a profound believer in vaccination and dieting of cattle for controlling Bang's disease. He also favors the agglutination test. The paper was discussed by Dr. W. E. Cotton, of Auburn, Ala., who probably has done as much laboratory work on this disease as any other one person. He expressed some splendid ideas concerning Bang's disease, but did not agree with Dr. Stewart concerning vaccination.

Following the discussion on Bang's disease, Dr. John S. Whitfield, Associate Professor of Veterinary Medicine, at State Col-

lege, gave a short talk and asked for the coöperation of the veterinarians in the state. He said that he would do the best he knew how to coöperate with the veterinarians and the Association.

At the afternoon session, in the absence of Dr. C. D. Stubbs, of Little Rock, Ark., Dr. McAdory delivered an address on "The Future of the Veterinary Profession." He enumerated many of the ups and downs of the profession and expressed the desire for more coöperation on the part of veterinarians generally. Dr. T. A. Sigler, of Greencastle, Ind., gave a very instructive talk on "Equine Diseases." His remarks dealt principally with diseases of the oral cavity. Dr. E. S. Brashier, of Jackson, conducted a round-table discussion on regulatory matters, with particular reference to the control and eradication of rabies.

A banquet was held in the Venetian Room of the Edwards Hotel, with Dr. Brashier acting as toastmaster. Following a delicious five-course dinner, Taylor's Studio of Dancing entertained members of the Association and guests with several dance numbers which began with a sailor's tap dance led by John Durr, six-year-old son of Dr. E. H. Durr, and ended with the Big Apple, danced by three couples. Following the dancing, Mr. Scott Dickson, of the Jackson *Daily News*, held the interest of the crowd for about 20 minutes with his feats of magic. Little Miss Martha Chadwick, accompanied by Miss Ann Chadwick, daughters of Dr. Hudson Chadwick, gave two delightful vocal selections. Dan Riley, champion negro skater of the world, demonstrated his ability. A number of the guests and members of the Association were called upon for after-dinner speeches.

Immediately following the banquet, the Association was called into executive session. Dr. John H. Gillmann, of Memphis, Tenn., asked for the endorsement of the Mississippi veterinarians of the plan to invite the American Veterinary Medical Association to meet in Memphis in 1939 or 1940. This was unanimously approved. Hattiesburg was selected for the 1939 meeting of the Mississippi Association.

The election of officers resulted as follows: President, Dr. E. S. Brashier, of Jackson; vice-president, Dr. J. T. Alston, of Tupelo, and secretary-treasurer, Dr. E. H. Durr (reëlected), of Jackson. Dr. R. H. Stewart, of Jackson, was reëlected delegate to the American Veterinary Medical Association House of Representatives, with Dr. O. M. Norton, of Greenville, selected as alternate.

A letter was read from Dr. W. W. Dimock, Lexington, Ky., relative to a special committee on periodic ophthalmia. The secretary

was instructed to write Dr. C. P. Fitch, of Saint Paul, Minn., to the effect that the Association heartily approved of the efforts of the A. V. M. A. to secure better recognition for veterinarians in public health work. Approval was given also to the plans for revamping the JOURNAL of the American Veterinary Medical Association and the employment of a field man as assistant secretary of the A. V. M. A., outlined in a letter writen by Dr. William Moore, member of the Executive Board of the A. V. M. A. for District 4.

E. H. DURR, *Secretary-Treasurer.*

NEVADA STATE VETERINARY ASSOCIATION

The annual meeting of the Nevada State Veterinary Association was held at the University of Nevada, Reno, January 28, 1938. Dr. W. K. Shidler, of Reno, presided.

Dr. H. H. Cole, of the College of Agriculture, University of California, was guest speaker. He gave two comprehensive discussions on the subject of hormones: "Regarding the General Nature of Hormones" and "Hormes Concerned with Reproduction."

Dr. R. A. Given, B. A. I. Inspector-in-Charge in Nevada, reported on the progress of the Bang's disease project at the afternoon session. This was followed by a general symposium on Bang's disease, and case reports made by practitioners and veterinarians in state and federal employ.

At the evening session, Dr. L. R. Vawter, of the University of Nevada, spoke on "Research in 1937." A discussion of plans for the control of equine encephalomyelitis in 1938 was led by Dr. Edward Records, of the University of Nevada.

Officers elected for the ensuing year are: President, Dr. L. R. Vawter; vice-president, Dr. Geo. E. Bamberger, and secretary-treasurer, Dr. Warren B. Earl, all of Reno. Dr. Wm. R. Smith, of Las Vegas, was elected delegate to the A. V. M. A. House of Representatives and Dr. L. R. Vawter was chosen as alternate.

WARREN B. EARL, *Secretary-Treasurer.*

VIRGINIA CONFERENCE FOR GRADUATE VETERINARIANS

The fifth annual Conference for Graduate Veterinarians was held at the Virginia Polytechnic Institute, Blacksburg, February 1-3, 1938. This conference was offered by the Department of

Biology, under the direction of Dr. I. D. Wilson, head of the Department, and his staff. Fifty-four veterinarians were registered.

The address of welcome was given by Prof. H. L. Rice, Dean of Agriculture, and then the following program was presented:

"Periodic Ophthalmia," by Dr. Lawrence O. Mott, U. S. Bureau of Animal Industry, Winchester.

"Swine Erysipelas," "Blood Transfusion in the Dog" and "Certain Feed-Lot Toxemias," by Dr. Ashe Lockhart, Kansas City, Mo.

"Differential Diagnosis and Diseases of Small Animals," by Dr. E. B. Dibbell, Baltimore, Md.

"Some New Developments in Veterinary Medicine," by Dr. Oscar V. Brumley, president of the American Veterinary Medical Association, Columbus, Ohio.

"Differential Diagnosis of Infectious Respiratory Diseases of Poultry," by Dr. E. P. Johnson, Blacksburg.

"Farm Forages and Nutritional Deficiencies," by Dr. A. D. Pratt, Blacksburg.

The clinic for large animals, with both lectures and demonstrations, was conducted by Drs. H. H. Adair, of Bristol, and H. T. Farmer, of Richmond. Interesting discussions of the various papers and addresses were conducted at the close of each period.

Dr. Brumley, in his eloquent manner, laid special emphasis on the need of higher standards for veterinary education. He also emphasized the importance of increasing the membership in the A. V. M. A. and pointed out the small percentage of membership in the A. V. M. A. as compared with other national professional associations.

Dr. Lockhart delivered a masterly lecture and demonstration on blood transfusion in the dog and his paper on "Certain Feed-Lot Toxemias" was considered a masterpiece by all of those who were fortunate enough to hear it. He brought out the fact that "Kojic" acid was produced and responsible for the toxemia produced by over-feeding. Dr. Dibbell's lecture and demonstration on small animals was very interesting.

At a business meeting of the Virginia State Veterinary Medical Association, held Tuesday evening, the resolution adopted by the A. V. M. A., at Omaha, in connection with the Social Security Act, was endorsed.

A banquet was held Wednesday evening in the Faculty Center, with 38 in attendance. Dr. E. P. Johnson acted as toastmaster and related a few hilarious jokes. He called on several of those present for impromptu addresses. During the banquet, Dr. Wilson read a bill that had been introduced into the Virginia Legislature designed to amend the Veterinary Practice Act. It was decided to call a special meeting of the State Association to consider this legislation, and a committee known as the Emer-

gency Legislative Committee was appointed by President O. F. Foley to go into action at once.

A. J. SIPOS, *Secretary.*

VETERINARY ASSOCIATION OF MANITOBA

The forty-eighth annual meeting of the Veterinary Association of Manitoba was held in the Royal Alexander Hotel, Winnipeg, on February 11, 1938. The meeting was attended by more than 50 veterinarians, many of whom were practitioners from country points.

Following the address of the president, Dr. J. W. Fasken, of Portage la Prairie, the balance of the morning session was taken up by a discussion of Association affairs. The report of Dr. W. Hilton, Secretary-Treasurer and Registrar, showed the Association to be in good financial condition.

The following officers were elected for 1938: President, Dr. F. M. Coombs, Hamiota; vice-president, Dr. J. A. Allen, and secretary-treasurer and registrar, Dr. W. Hilton, both of Winnipeg. Dr. R. McEwen, of Stonewall; Dr. A. Savage and Dr. R. H. Lay, both of Winnipeg, and Dr. D. J. Lawson, of Shoal Lake, were elected to the Council.

Owing to the unavoidable absence of the newly elected president, Dr. Coombs, Dr. J. A. Allen acted as chairman during the afternoon session, which was opened with an address by Dr. A. Savage, of the Manitoba Department of Agriculture, on "Equine Encephalomyelitis." This was received with a great deal of interest and the general discussion that followed brought out some interesting phases of the treatment of this malady.

Dr. J. K. Morrow, of Winnipeg, gave an address on "Specialization." This was followed by a discussion, led by Dr. A. L. Alton, of Portage la Prairie, of a number of interesting case reports and problems confronting the veterinarian in the control of Bang's disease. Dr. H. J. Tingley's paper, "Amniotic Extract and Mange," gave some interesting side lights on the treatment of mange in canines and felines.

Following the adjournment of the meeting, Dr. Morrow presented some motion-pictures showing clinical cases of equine encephalomyelitis.

An item of interest to the practitioners present was the report given by Dr. Savage on the progress being made in the organization of a laboratory at Winnipeg. by the Manitoba Department of Agriculture. Dr. Savage will be in charge of this unit and

will undertake to render laboratory diagnostic service and conduct research work in animal diseases.

R. H. LAY, *Reporter.*

ILLINOIS STATE VETERINARY MEDICAL ASSOCIATION

The fifty-sixth annual meeting of the Illinois State Veterinary Medical Association was held in Springfield, at the Hotel Abraham Lincoln, February 17-18, 1938.

Dr. G. W. Jensen, of Antioch, in his presidential address, recommended that the Association approve the action of the Executive Board in asking the University of Illinois to establish a college of veterinary medicine in connection with the institution.

The morning and afternoon sessions of both days of the meeting were taken up by the literary program. The following papers were presented:

"My Experiences in Treating Milk Fever and Results Obtained," by Dr. W. W. Warnock, Aledo.

"Sheep Diseases and Feeding Problems," by Dr. L. D. Frederick, Chicago.

"The Feeding and Care of Brood Sows and Litters," by Dr. W. B. Massie, Boston.

Address by Dr. H. C. Rinehart, Chief Veterinarian, Springfield.

"The Newer Conception in Our Knowledge of Virus Diseases," by Dr. A. Eichhorn, Pearl River, N. Y.

"The Seventh World's Poultry Congress," by Mr. R. V. Usrey, Superintendent, Division of Poultry Husbandry, Department of Agriculture, Springfield.

"An Animal-Health Program for Low-Income Farmers," by Mr. I. F. Gillmor, State, Community and Coöperative Specialist, U. S. Department of Agriculture, Farm Security Administration, Champaign.

"Pertinent Swine Problems," by Dr. J. D. Ray, Omaha, Neb.

"Random Remarks Relating to Selected Subjects and Their Treatment (Small Animals)," by Dr. A. R. Theobald, Cincinnati, Ohio.

"Skin Diseases Commonly Seen in Small-Animal Practice," by Dr. O. Norling-Christensen, Wilmette.

"Equine Encephalomyelitis," by Dr. Frank Breed, Lincoln, Neb.

"Treatment of Encephalomyelitis," by Dr. R. S. Gerard, Sigourney, Iowa.

"Swine Erysipelas," by Dr. C. M. Rodgers, Avon.

The business session was held the afternoon of February 17. Officers were elected as follows: President, Dr. C. D. Dille, of Cairo; vice-president, Dr. W. B. Holmes, of Springfield, and member of the Executive Board, Dr. C. Vanderwarf, of Chicago Heights.

A resolution was passed approving the recent action of the American Veterinary Medical Association in calling to the attention of the Public Health Service of the United States Treasury Department that the qualifications of the veterinarian were completely overlooked or ignored in the Public Health Program under Title VI of the Social Security Act.

At the dinner session, Thursday evening, Dr. L. A. Merillat, of Chicago, was presented with an engraved watch and chain as a gift from his many friends in the veterinary profession in Illinois, to commemorate his fifty active years of untiring service to the veterinary profession of this country. Accompanying the gift was a plaque signed by the friends who participated in honoring Dr. Merillat.

C. C. HASTINGS, *Secretary.*

MIDWEST SMALL ANIMAL ASSOCIATION

Through the efforts of Drs. H. G. Dow, of Fort Madison, Iowa; J. H. Krichel, of Keokuk, Iowa, and Dr. R. V. Beard, of Burlington, Iowa, there was organized, at Burlington, on February 24, 1938, the Midwest Small Animal Association. There were 29 charter members from Iowa, Illinois and Missouri, all general practitioners, who wish to know more about the diseases of small animals. Dues were fixed at $3.00 per year.

The following papers were preesnted:

"Epileptiform Convulsions," by Dr. C. M. Collins, Ottumwa, Iowa.

"Sulfanilamide in Small-Animal Practice," by Dr. F. J. Crow, Iowa City, Iowa.

"Drainage of the Ossa Bulla," by Dr. H. R. Hornbaker, Galesburg, Ill.

Dr. C. L. McGinnis, of Peoria, Ill., gave a demonstration of a complete clinical examination. It is planned to hold a small-animal clinic, at Burlington, in May or June.

C. L. MCGINNIS, *Secretary-Treasurer.*

Friends are not so easily made as kept.

—*Lord Halifax*

The true university of these days is a collection of books.

—*Carlyle*

NECROLOGY

RUDOLPH RAY BOLTON

Dr. R. R. Bolton, of Newburgh, N. Y., died on September 5, 1937, following a cerebral hemorrhage.

Born at Mendon, Ohio, April 20, 1884, Dr. Bolton attended Ohio State University. Later he entered Cornell University, where he completed the course in veterinary medicine in 1912. From February, 1914, to April, 1918, he taught medicine and physical diagnosis in the Division of Veterinary Medicine, Iowa State College. More recently, he had been employed by Sheffield Farms Company, of New York City.

Dr. Bolton joined the A.V.M.A. in 1922.

FREEMAN ALFRED MARSHALL

Dr. Freeman A. Marshall died suddenly of a heart attack at his home in Indiana, Pa., October 8, 1937.

Born near Rome, Pa., February 24, 1874, Dr. Marshall obtained his preliminary education in public schools. He then entered the Indiana Veterinary College and was graduated with the class of 1906. He engaged in private practice for a short time and then accepted a position at the Annadean Farm, near Cleveland, Ohio. In 1918, he entered the service of the Pennsylvania Bureau of Animal Industry and continued in this work until his death. He was a pioneer in sterility work with cattle and was unusually well informed on Bang's disease. He did much to popularize the Pennsylvania Plan for the control of Bang's disease, and there were few who could equal or surpass his technic in surgery.

Dr. Marshall joined the A.V.M.A. in 1916. He was resident secretary for Pennsylvania, 1929-1930. He was a member of the Twelfth International Veterinary Congress and a member and past master of Roman Lodge No. 418, F. & A. M. He is survived by his widow, one son, one daughter and two brothers, Drs. C. J. Marshall (U. P. '94), and Leslie G. Marshall (U. P. '04), both of Philadelphia.

The Commonwealth of Pennsylvania has lost an outstanding citizen; the Pennsylvania Bureau of Animal Industry, a faithful, efficient and loyal employé, and the veterinary profession, a practioner of unquestionable merit. Dr. Marshall's congenial and affable disposition, his practical knowledge of veterinary subjects and his unexcelled diagnostic ability endeared him to all.

H. M. K.

HENRY NUNN

Dr. Henry Nunn died at the Oregon State Hospital, Salem, on January 9, 1938. He had been a patient at the institution since May 13, 1919, when he suffered a nervous breakdown. He had been more or less an invalid from that time until his death.

Born in Ontario, Canada, in 1872, Dr. Nunn was graduated from the Ontario Veterinary College in 1893. He located at McMinnville, Ore., and saw service during the Spanish-American War. Later he returned to McMinnville and practiced there for a number of years. During 1915 and 1916, he served as Assistant State Veterinarian to the Oregon State Live Stock Sanitary Board. He then returned to McMinnville again and practiced for about three years, when his health failed.

Dr. Nunn joined the A.V.M.A. in 1904. He was one of the first members of the Oregon Veterinary Medical Association and was always an ardent supporter of higher education. He held a life membership in the Masonic order in Canada. Dr. Nunn was a lover of horses and was possessed of a keen sense of Irish humor. He was respected by all with whom he came in contact.

W. H. L.

ERNEST N. HUTCHINSON

Dr. Ernest N. Hutchinson, of Olympia, Wash., died in a Seattle hospital on January 30, 1938, at the age of 73 years. He had been Secretary of State since 1932.

Born in Milford, N. Y., Dr. Hutchinson attended public schools and then decided to "go West." He drove a stage-coach in the Black Hills of South Dakota and rode the range in the Texas Panhandle before turning to veterinary medicine.

Dr. Hutchinson was graduated from the Chicago Veterinary College in 1894 and was appointed an assistant inspector in the U. S. Bureau of Animal Industry, August 15, 1896. The following year he was placed in charge of the B.A.I. station at Portland,

Ore. On May 1, 1900, he was promoted with the rank of inspector-in-charge and placed in charge of the stations at Tacoma and Seattle, Wash., in addition to Portland. During the summer of 1903, Dr. Hutchinson was transferred to Salt Lake City, Utah, to conduct work on the eradication of sheep scabies. He was transferred to the field station at Pendleton, Ore., early in 1907 and, on July 1, 1909, was made inspector-in-charge at Portland. After several assignments to inspection work along the Canadian border, over a period of about ten years, Dr. Hutchinson left the service (June 30, 1921), to become identified with the dairy interests of the State of Washington. For a number of years, Dr. Hutchinson was a field representative of the Washington Dairy Products Marketing Bureau, which position he held at the time of his election to the office of Secretary of State.

Dr. Hutchinson was in the midst of a campaign to develop dairying when he was asked to enter the race for Secretary of State in 1932. Within two hours of the closing time for filing, he said, "Such a thing as seeking public office never entered my head." He carried every county in the primaries and in the general election he defeated his rival candidate by 77,000 votes. Although he refused to campaign for reëlection in 1936, he polled 127,191 votes in the primary, more than the combined total of his two opponents. In the general election he won by 175,574 votes.

Surviving Dr. Hutchinson are his widow, one son and one stepdaughter.

STEPHEN O'TOOLE

Dr. Stephen O'Toole, of Sacramento, Calif., died on February 3, 1938, at San Diego, Calif. His death was attributed to suicide, following a serious illness of several years. Born in Shawnee, Ohio, December 19, 1878, Dr. O'Toole attended local schools. During his later boyhood years, he worked in a mine and then clerked in a mercantile store. He entered the College of Veterinary Medicine, Ohio State University and was graduated in 1908.

For a few months after graduation, Dr. O'Toole was employed as a veterinary inspector by the U. S. Bureau of Animal Industry and then was transferred to the Philippine Islands, where he served until 1911. After returning to the States, he took postgraduate work in pathology and bacteriology for 18 months at the Harvard Medical School, Boston. The next three years, he was engaged in teaching and research work at the North Dakota

Agricultural College and Experiment Station at Fargo, and then, from 1916 to 1919, he was Chief Food Inspector, City of Atlanta, Ga. In 1920, he was employed by the Division of Animal Industry, California Department of Agriculture, where he served as bacteriologist, supervising veterinarian in tuberculosis eradication work and, for the final five months, in charge of the Division's poultry pathological laboratory at San Diego.

Dr. O'Toole joined the A. V. M. A. in 1913 and was a member of the California State Veterinary Medical Association. He resigned from both organizations in 1937 on account of ill health. He was a charter member of Alpha Chapter of Alpha Psi Fraternity. He is survived by his widow, one son and one sister.

C. V. D.

CHARLES HINKLE ELLIS

Dr. Charles H. Ellis, of La Center, Ky., died at his home February 1, 1938, following a short illness. He had suffered a stroke of paralysis about two weeks before his death.

Born in Hinkleville, Ky., December 1, 1879, Dr. Ellis attended local grade and high schools before entering the Terre Haute Veterinary College. Following his graduation in 1913, he returned to Kentucky and located at Barlow for general practice. Later he moved to Lovelaceville, where he remained until about a year ago, when he removed to La Center. He was Veterinary Inspector for Ballard County.

Dr. Ellis joined the A. V. M. A. in 1923. He is survived by his widow, three children by his first marriage, three brothers and three sisters.

CAPT. PATRICK H. HUDGINS

Capt. Patrick H. Hudgins, U. S. Army, retired, died at Carmel, Cal., February 8, 1938. His retirement, because of disability received in service, dated from July 2, 1928.

Born in Richmond, Va., January 15, 1887, Capt. Hudgins obtained his veterinary training at the Ontario Veterinary College. He was graduated in 1910 and six years later began his military service as a 2nd lieutenant, Veterinary Corps, Virginia National Guard. He was promoted to 1st lieutenant, Veterinary Corps, National Army, on March 18, 1918, and was promoted to the rank of captain while with the A. E. F. He entered service

in the Regular Army on July 1, 1920, as a 2nd lieutenant and reached the grade of captain on April 6, 1924.

Capt. Hudgins joined the A.V.M.A. in 1910 and resigned in 1928, when he was retired from the army. R. J. F.

HARRY BANNISTER

Dr. Harry Bannister, of Tampa, Fla., died on the 75th anniversary of his birth, February 11, 1938.

Born in Bradford, England, Dr. Bannister received his veterinary education at the University of Pennsylvania. Following his graduation in 1890, he conducted a general practice in Roanoke, Va., for nearly 30 years. He retired from active practice about 20 years ago and had lived in Tampa during this time.

Dr. Bannister joined the A.V.M.A. in 1908. For many years he was a member of the Virginia State Veterinary Medical Association and served a term as president. He also was a member of the Virginia State Board of Veterinary Examiners and for a number of years served as secretary-treasurer. He is survived by his widow and one daughter.

JOSEPH HENRY GILL

Dr. Joseph H. Gill, of Portsmouth, Ohio, died suddenly of a heart attack, February 12, 1938, shortly after making a professional call.

Born in Cincinnati, Ohio, February 25, 1878, Dr. Gill attended grade and high schools and Spring Institute before entering the Cincinnati Veterinary College. Following his graduation in 1910, he entered general practice at Portsmouth and remained there until his death.

Dr. Gill joined the A.V.M.A. in 1920. He was a member of the Ohio State Veterinary Medical Association. He is survived by his widow (née Mary Weismann), two daughters, two sons and four brothers.

WILLIAM B. CRAIG

Dr. William B. Craig, of Indianapolis, Ind., died in Saint Vincent's Hospital, February 16, 1938, after an illness of more than two months. He underwent an operation in December and was taken home. Later his condition became so serious that he was returned to the hospital on January 19.

Born in Ayrshire, Scotland, November 5, 1868, Dr. Craig came to the United States when he was 13 years old and located in Greenwood, Ind. He was graduated from the Ontario Veterinary College in 1889 and four years later was graduated from the Indiana Medical College (now part of University of Indiana). He entered practice in Indianapolis and later became identified with the Indiana Veterinary College, which was organized in 1892. He served as professor of anatomy, dentistry and operative surgery for a number of years and also as dean of the faculty from 1900 until the institution closed its doors in 1924.

Dr. Craig joined the A. V. M. A. in 1904. He served as a member (1907-08) and as chairman (1910-11) of the Special Committee on Revision of the Constitution and By-laws; as a member of the Committee on Local Arrangements for the Indianapolis meeting in 1912; as a member of the Committee on Intelligence and Education (1912-15); as a member of the Committee on Necrology (1921-22), and as resident secretary for Indiana (1921-22). He had been secretary-treasurer for the Indiana Veterinary Medical Association for a number of years.

The survivors include his widow (née Madge Roquet), three daughters, one son and two sisters.

HARRY E. BATES

Dr. Harry E. Bates, of South Norwalk, Conn., died at his home on February 17, 1938. He was a graduate of the American Veterinary College, class of 1889, and had practiced in South Norwalk for 45 years.

Dr. Bates joined the A. V. M. A. in 1890 and served as Resident Secretary for Connecticut from 1892 to 1894. He was a member of the Committee on Local Arrangements for the New Haven meeting in 1906. He was a member of the Connecticut Veterinary Medical Association and served as president in 1904. He was a member of the State Board of Veterinary Registration and Examination from 1913 to 1923, and during seven years of this period he served as secretary-treasurer. He was a member of Trinity Episcopal Church, Old Well Lodge, F. and A. M., and the Shrine.

Surviving Dr. Bates are his widow, one daughter and two stepdaughters.

G. E. C.

PAYSON E. SCHWIN

Dr. Payson E. Schwin, of Elkhart, Ind., was killed in an automobile accident on February 19, 1938. While he was making a professional call near Kessington, Michigan, in a blinding snow storm, his car collided with a truck on U. S. 112. He died shortly after the accident.

Born near Middlebury, Ind., December 2, 1865, Dr. Schwin entered the Ontario Veterinary College at the age of 20. He was graduated with the class of 1888. He received the gold medal for the highest average in his examinations. He was the first American student to win this honor. Soon after graduation, Dr. Schwin located in Elkhart, where he practiced until his death. He was prominent in public affairs and had a wide circle of acquaintances.

Dr. J. L. Axby, state veterinarian of Indiana, paid Dr. Schwin the following tribute:

> No veterinarian was ever more highly esteemed or held in higher regard than Dr. Schwin; because he was always ready and willing to render assistance in service or money to every worthwhile project that would elevate the profession or enhance the live stock industry. He took advantage of every opportunity for advancement, was an extensive reader, kept himself in the vanguard of veterinary progress, and was always active in his state association.
>
> He was possessed with a practical idealism which his service and conduct clearly demonstrated; thus all who knew him loved him and his reputation for honesty, uprightness and ability far exceeded his circle of direct contacts.

Dr. Schwin joined the A.V.M.A. in 1911. He was a member of the Elkhart Kiwanis Club, the Fairview Grange and the Congregational Church. He is survived by his widow (née Edith Bodge), one daughter, one son and two sisters.

F. C. T.

FREDERICK C. JONES

Dr. Frederick C. Jones, of Vancouver, British Columbia, Canada, died on February 20, 1938, in his 69th year.

Born at Brownedge, Staffordshire, England, Dr. Jones came to Canada in 1897. The fall of that year, he entered the Ontario Veterinary College. Following his graduation in 1899, he practiced at Fenwick, Ont., for nine years, and then entered the service of the Health of Animals Branch, Dominion Department of Agriculture, at Toronto. The following year he was transferred to Winnipeg and, six years later, to Vancouver. He was

in charge of the administration of the Meat and Canned Foods Act in British Columbia for 17 years. He was superannuated in 1933.

Dr. Jones joined the A. V. M. A. in 1913. He is survived by his widow (née Sara Ann Barwell), two sons and a daughter. One son is Dr. Frederick Oulton Jones (Ont. '31), of Vancouver.

J. G. J.

HARRY D. LARZELERE

Dr. Harry D. Larzelere, of Janesville, Wis., died at his home on February 20, 1938. He had been in failing health for several months.

Born at Roseville, Ohio, October 10, 1883, Dr. Larzelere attended local grade and high schools before entering the Ohio State University for the study of veterinary medicine. He was graduated with the class of 1909, and shortly thereafter entered the service of the U. S. Bureau of Animal Industry. During recent years he had been stationed at various points in Wisconsin on hog cholera control.

Dr. Larzelere joined the A.V.M.A. in 1921. He was a member of the Wisconsin Veterinary Medical Association and National Association of B.A.I. Veterinarians. He was a member of the Masonic lodge of Eau Claire, Wis., and of the Janesville Chapter No. 69, Order of the Eastern Star. He is survived by his widow (née Ina Sprung), one son, his mother, one brother and two sisters.

Dr. Larzelere was possessed of exceptional ability and had the confidence of all federal and state officials, as well as the practicing veterinarians of Wisconsin, who did not hesitate to call upon him for help whenever needed. They feel that they have suffered a distinct loss in the death of their esteemed colleague.

J. S. H.

ALEXANDER M. McKAY

Dr. Alexander M. McKay, of Calgary, Alberta, Canada, passed away on February 21, 1938, following a brief illness due to angina pectoris. Although his health had commenced to fail about two years ago, it was not until two days prior to his death that anything serious was known to exist.

Born at Morewood, Ont., on February 27, 1873, Dr. McKay was graduated from the Ontario Veterinary College in 1893 and practiced his chosen profession at Winchester, Ont., for 13 years. In 1906, he joined the Health of Animals Branch, Dominion Department of Agriculture, and was attached to the Contagious Diseases Division at Calgary for eight years. He rendered valuable service in connection with the campaign then being waged against dourine and glanders in the southern part of the province.

The late Dr. J. G. Rutherford was at this time in charge of the Animal Husbandry Branch of the Department of Natural Resources of the Canadian-Pacific Railway at Calgary, and he persuaded Dr. McKay to accept a position with him. Dr. McKay held this position until 1917, when he resigned to reënter private practice with the late Dr. T. E. LeClaire. They conducted a successful practice together until 1920, when Dr. LeClaire was forced to retire on account of ill health. Dr. McKay carried on alone until 1924, when he sold a half interest in the practice to Dr. J. G. Anderson, who purchased the practice outright the following year. Since that time Dr. McKay had devoted his time to building up a choice herd of Jersey cattle.

Dr. McKay joined the A. V. M. A. in 1909. He is survived by his widow (neé Molly McVeigh), one daughter and one son.

J. C. H.

GEORGE M. OLDS

Dr. George M. Olds, of Hartford, Mich., died at his home on February 24, 1938, after an illness of several months, caused by asthma and cardiac disease.

Born in Hartford, November 27, 1860, Dr. Olds engaged in farming until he was about 40 years of age. He then decided to study veterinary medicine and entered the Grand Rapids Veterinary College. Following his graduation in 1901, he entered general practice at Hartford and had a large clientele throughout Van Buren County.

When the Hartford Fair Grounds were built, in 1913, Dr. Olds supervised the construction of the race track. For many years he was prominently identified with county fairs and race meets in both Michigan and Indiana. He is survived by his widow (née Annabelle Shaw), three sons, two daughters and two brothers.

ARTHUR JOHN ERICKSON

Dr. A. J. Erickson, of Geneseo, Ill., died at his home on February 24, 1938. He had been ill of coronary thrombosis since December and had been bedfast for four weeks.

Born in Stetsonville, Wis., February 28, 1881, Dr. Erickson attended local schools and the University of Wisconsin before entering the Chicago Veterinary College. He was graduated in 1911 and entered practice at Donovan, Ill. He remained there until 1927, when he went to Tuscola, as Douglas County Veterinarian, engaged in tuberculosis eradication. Two years later, he removed to Toulon, where he engaged in the same work for Stark County. In 1930, he again made a change, going to Geneseo, where he was employed by the State Department of Agriculture, supervising tuberculosis eradication in Henry, Bureau, Rock Island, Carroll and Jo Daviess counties.

Dr. Erickson joined the A. V. M. A. in 1929. He was a member of the Illinois State Veterinary Medical Association. Fraternally he was identified with the Masons and the Modern Woodmen of America. He is survived by his widow (née Nellie Hodgden), one daughter, one son, four brothers and two sisters.

SAMUEL BROAD

Dr. Samuel Broad, of Chesterton, Ind., died in a Logansport, Ind., hospital on February 26, 1938, as the result of injuries he received in an automobile accident on June 7, 1937.

Born in Marijosa, Ontario, Canada, February 24, 1862, Dr. Broad was graduated from the Ontario Veterinary College in 1890. He practiced first at Crown Point, Ind., and later at Momence, Ill. In 1908, he moved to Evanston, Ill., and three years later to Chesterton, Ind., where he conducted a general practice. He was making a professional call, the evening of June 7, when his car was struck by a truck. He had been a hospital patient ever since.

Surviving Dr. Broad are one daughter, one son, two sisters and three brothers.

CHARLES O. KROENER

Dr. C. O. Kroener, well-known Illinois veterinarian, died at the Mercy Hospital, Chicago, February 26, 1938, following an operation for cancer of the stomach. As he had worked without

known interruption up to the time of going to the hospital, his death was unexpected even among his closest friends.

Born at Evansville, Indiana, September 10, 1889, Dr. Kroener came to Chicago with his family in 1908. His mother, a widow of outstanding motherly instincts, decided to move to Chicago in order to give her son an opportunity to work his way through the Chicago Veterinary College. He obtained a position under the shipping clerk of the Boston Store and at night did clerical work, besides making himself generally useful at the hospital of Wright and Merillat, where he remained as a student employé throughout his college course and as a trusted graduate assistant for 14 years, following his graduation in 1911.

Following his separation from the hospital of Wright and Merillat, Dr. Kroener began to build up a clientele of his own, at the same time holding several subsidiary positions of a political nature. For a number of years, he was a member of the Illinois State Board of Veterinary Medical Examiners; an assistant state veterinarian, veterinarian for several public institutions of Cook County, and later milk inspector in the Chicago Department of Health. At the time of his death, he held an important full-time position in the local public health service.

Dr. Kroener joined the A.V.M.A. in 1917. Evidence of his beautiful Spencerian penmanship may be found on some of the records of the A.V.M.A. for 1916-1917, while this writer was its secretary and the Association's income did not permit the employment of extra clerical help.

Kroener, as he was called in his inner circle of friends, was a hard, tireless worker, a man of force, who won the confidence of his associates by having confidence in himself—a widow's son who made good in spite of his boyhood handicaps.

He is survived by his widow (née Anna Quick), two charming daughters and two stalwart sons, all tutored and educated, ready to face the world through the sacrifices of their devoted parents. Extolling the virtues of an employé of 16 continuous years is scant courtesy to his memory, the tenure is the more revealing eulogy.

L. A. M.

JAMES FLEMING

Dr. James Fleming died at his home in Rochelle Park, N. J., on March 3, 1938, at the age of 71 years. He had been in failing health before and since his retirement from the U. S. Bureau of Animal Industry, on September 30, 1936.

Born in Canada, Dr. Fleming attended grade and high schools before entering the Ontario Veterinary College. Following his graduation in 1890, he located in Detroit, Michigan, and practiced there for several years. In 1896, he entered the federal service and was assigned to meat inspection at Chicago. He was promoted to assistant inspector-in-charge while in Chicago and, several years later, was transferred to Fort Worth, Texas, as inspector-in-charge. A little later, he was transferred to Kansas City, Kan., in the same capacity and his last assignment, in 1928, was at Clifton, N. J., where he was in charge of the U. S. Animal Quarantine Station. While in Chicago, Dr. Fleming took a course in human medicine at the Harvey Medical College and received his M. D. in 1903.

Dr. Fleming joined the A. V. M. A. in 1916. He was a member of the National Association of B. A. I. Veterinarians and served as president in 1922 and 1923. He was a member of the Twelfth International Veterinary Congress. He is survived by his widow and one son.

JOHN AUGUST ANDERSON

Dr. John A. Anderson, of Lake Park, Minn., died on March 1, 1938, as the result of a broken neck and other injuries sustained when his car was hit by another in a heavy fog. The accident happened in front of his home on Highway 10 and death was almost instantaneous.

Born near Lake Park, January 31, 1879, Dr. Anderson was a graduate of the Chicago Veterinary College, class of 1905. He practiced at Lake Park ever since his graduation, except for a period of about two years when he was located at Fertile, Minn. He is survived by four sisters and two brothers.

J. P. F.

D. JUDSON CARGILL

Dr. D. Judson Cargill, of Bellefontaine, Ohio, died at his home on March 7, 1938, at the age of 78 years, after a protracted illness. He had been in poor health for a number of years, having suffered from diabetes. He was a graduate of the Ohio Veterinary College, of Cincinnati, class of 1896. He had combined his veterinary practice with farming and stock-raising for many years.

The deceased was the son of Henry Cargill, a non-graduate practitioner who died in 1916. The survivors of Dr. Judson

Cargill include his widow (née Edith Fullerton) and a half brother, Dr. Raymond H. Cargill (Cin. '11), of Troy, Ohio.

LIONEL LEE STIER

Dr. Lionel L. Stier, of Grand Mound, Iowa, passed away at the Mercy Hospital, Clinton, Iowa, on March 5, 1938, after a brief illness. He was injured in an automobile accident last August and was a patient in the Mercy Hospital for several months. In November, he went to the U. S. Veterans Hospital in Des Moines, where he spent several months.

Born in Grand Mound, August 20, 1891, Dr. Stier was graduated from the Chicago Veterinary College in 1915. He was commissioned as a second lieutenant in the Veterinary Corps of the Army, March 3, 1918, and a few days later was directed to report for duty with the 34th Division at Camp Cody, N. Mex. Later he was assigned to the 67th Infantry Brigade, A. E. F., and was discharged September 3, 1919. Surviving Dr. Stier are two brothers.

H. E. T.

J. OMER GREESON

Dr. J. O. Greeson, of Kokomo, Ind., died in a hospital at Kissimmee, Fla., March 8, 1938, following a cerebral hemorrhage which overtook him about a week previously. Dr. Greeson maintained a winter home at Saint Cloud, Fla., and had spent his winters there for the past several years.

Born in Morgan County, Ind., August 12, 1856, Dr. Greeson spent his boyhood on the family farm and attended local schools. He then entered the University of Pennsylvania for the study of veterinary medicine and was graduated with the class of 1893. Subsequently, he studied for a year at Johns Hopkins University in Baltimore. He practiced for many years in Howard, Tipton and Cass counties, Ind., with marked success. He is said to have been the first graduate veterinarian to locate in Howard County.

Dr. Greeson was a member of the A. V. M. A. from 1893 until 1921. He took quite an interest in local civic affairs, was quite a Bible student, and was a charter member of the I. O. O. F. Lodge at Alto. Dr. Greeson never married. He is survived by one brother, three sisters-in-law, five nieces and one nephew.

FRED H. HASENMILLER

Dr. Fred H. Hasenmiller, of Davenport, Iowa, died in the Mercy Hospital, that city, on March 10, 1938, as the result of injuries received in a fall a few days previously, while performing his duties as Municipal Meat and Dairy Inspector.

Born in Davenport, May 10, 1878, Dr. Hasenmiller attended local schools and took a business course before deciding to study veterinary medicine. He attended Iowa State College for two years, but finished his veterinary education at the Chicago Veterinary College in 1903. He practiced in Davenport for a number of years, then moved to Eldridge, Iowa. He returned to Davenport about nine years ago to accept the position which he held until the time of his death

Dr. Hasenmiller joined the A. V. M. A. in 1918. He was one of the founders of the Eastern Iowa Veterinary Association. He was a member of Battery B of Davenport, and served in the Mexican uprising. He was commissioned as a second lieutenant in the Veterinary Corps of the Army during the World War and spent several months on active duty at the Veterinary Training School at Camp Lee, Va.

Surviving are the widow (neé Cary Denker), two daughters, one sister and three brothers.

CLYDE RAYMOND PETERSON

Dr. Clyde R. Peterson, of Chatsworth, Ill., was found dead in his office on March 13, 1938. The coroner returned a verdict of death from heart disease.

Born on a farm near Roberts, Ill., September 17, 1895, Dr. Peterson received his early education in the rural schools and then attended the Chicago Veterinary College for two terms (1915-16). He was inducted into military service at Camp Grant on October 3, 1917, and saw service overseas with Company H, 127th Infantry, as a part of the 32nd Division. He was gassed at Sesgy on July 30, 1918, and a few days after leaving the hospital was severely wounded by shrapnel when serving in the Meuse-Argonne engagement. He was still in the hospital at the time the Armistice was signed. After partially regaining his health, he resumed his veterinary studies at Michigan State College and was graduated with the class of 1924. He located at Chatsworth for general practice and remained there continu-

ously except for short periods spent in the U. S. Veterans' Hospital at Maywood, Ill.

Surviving Dr. Peterson are one daughter, his parents, three sisters and two brothers. He married Miss Leola Voss, of Lansing, Mich., in August, 1922. She died on September 17, 1928.

JOHN H. SCHRADER

Dr. J. H. Schrader, of Oxford, Iowa, died at the Mercy Hospital, Iowa City, Iowa, March 14, 1938, after putting up a valiant but losing fight against pneumonia.

Born at Greenfield, Iowa, September 19, 1894, Dr. Schrader attended grade and high schools before entering the McKillip Veterinary College. Following his graduation in 1918, Dr. Schrader spent a short time in the service of the U. S. Bureau of Animal Industry on meat inspection. He located at Oxford in 1920 and built up an extensive practice in that vicinity.

Dr. Schrader joined the A. V. M. A. in 1924. He was a member of the Eastern Iowa Veterinary Association and served one term as vice-president. He was a member of the Iowa Veterinary Medical Association and was active in the affairs of the Oxford Post of the American Legion. He took a very active interest in civic affairs and was an unsuccessful candidate for mayor of Oxford in 1928, but was elected to the city council two years later by large vote and had been reëlected each time since. In the summer of 1931, he was elected to the Oxford School Board, a post he continued to hold until his death. He was commissioned as a first lieutenant in the Veterinary Reserve Corps.

Surviving Dr. Schrader are his widow (née Hazel Egan), two daughters, his mother, one brother and four sisters.

PERSONALS

MARRIAGES

Dr. Carl M. Oldham (O. S. U. '37), of Dayton, Ohio, to Miss Mary Leaming, of Xenia, Ohio, January 28, 1938.

Dr. M. J. Bramley (O. S. U. '33), of Cleveland, Ohio, to Miss Ethel Paddon, of Youngstown, Ohio, at Youngstown, January 20, 1938.

BIRTHS

To Dr. and Mrs. Karl H. Willers, of Vancouver, Wash., a son, William, January 6, 1938.

To Dr. and Mrs. John Smith, of Stuttgart, Ark., a son, John William, December 14, 1937.

PERSONALS

DR. J. J. ARNOLD (Mich. '37) has located at Perry, Mich. for general practice.

DR. E. J. WALTERS (Gr. Rap. '06), of Wyandotte, Mich., spent the winter in Florida.

DR. W. L. TANNER (O. S. U. '32), of Van Wert, Ohio, is constructing a new veterinary hospital.

DR. W. E. BROWN (Iowa '37), has changed locations from Ankeny, Iowa to Kingsley, same state.

DR. LOUIS A. SORDO (A. P. I. '36), gives a new address: 6909 Wisconsin Ave. N. W., Bethesda, Md.

DR. C. E. LARSEN (St. Jos. '19), who has been in practice at Tilden, Neb., has removed to Rushville, Ind.

DR. S. N. MYERS (K. C. V. C. '13), has requested a change of address from Richfield, Utah, to 43 Van Wagenen St., Jersey City, N. J.

DR. PEYTON D. ATKINS (O. S. U. '04), of Hallsville, Ohio, had his left leg broken in an automobile accident the latter part of February.

DR. L. B. SWINGLEY (Chi. '09), of Oregon, Ill., has been reappointed Ogle County Veterinarian for another year, by the Board of Supervisors.

DR. W. T. BRINKER (O. S. U. '30), of Miamisburg, Ohio, recently purchased a short-wave diathermy machine for use in his practice.

DR. B. G. EATON (Ala. '36), of Delphos, Ohio, has removed to Fort Loramie, Ohio, and taken over the practice of the late Dr. N. F. Raterman.

DR. SIDNEY W. STILES (U. P. '32), of Falmouth Foreside, Maine, has fully recovered from his attack of undulant fever and has resumed practice.

DR. C. L. HAUPERT (O. S. U. '35), formerly of New Philadelphia, Ohio, is now with the Norden Laboratories, 244 West Fourth Ave., Columbus, Ohio.

DR. F. H. CONOVER (Ont. '34), formerly located at Petersburg, Ill., has opened the Conover Animal Hospital at 701½ North Evans Street, Bloomington, Ill.

DR. S. C. SORENSON (St. Jos. '13), of Tescott, Kan., has left the service of the U. S. Bureau of Animal Industry and is now located in Maywood, Calif.

DR. B. D. ROBBINS (Mich. '19), formerly of Holly, Mich., is now located in Cadillac, Mich., where he has taken over the practice of the late Dr. D. W. Curtis.

DR. J. L. WRIGHT (U. P. '32), of Lagrange, Ga., was unanimously elected City Meat and Milk Inspector at the meeting of the City Council on January 10.

DR. ROLAND SCOTT (Mich. '36), of Colon, Mich., has accepted a position on the staff of the Department of Veterinary Science, Oregon State College, at Corvallis.

DR. DONALD R. MORGAN (Wash. '30), who was on active duty in the Veterinary Reserve Corps, has accepted a position with the Washington State Department of Agriculture.

DR. HERBERT L. PARKER (Ind. '02), of Danville, Ill., has announced his candidacy for the Republican nomination for County Recorder, subject to the May 3 primary election.

DR. H. RASMUSSEN (McK. '13), of Balaton, Minn., breeds high-class registered Percheons and Holsteins. All of his horses are black and of the blocky type, Calypso line-bred.

DR. OLIVER H. LEONHART (Chi. '00) was retired from active service in the U. S. Bureau of Animal Industry, on December 31, 1937. He was on meat inspection at Oklahoma City, Okla.

DR. J. R. BROWN (O. S. U. '20), of Ottawa, Ill., addressed the local Rotary Club on February 2. His subject was "The Eradication of Bovine Tuberculosis from LaSalle County Herds."

DR. C. C. HASTINGS (Chi. '13), of Williamsville, Ill., was the guest speaker at the Farmers Evening School at Greenview, Ill., on March 21. His subject was "Malnutrition in Live Stock."

DR. C. E. WATSON (K. S. C. '35) has resigned from the U. S. Bureau of Animal Industry and is now associated with Dr. H. J. Hearrington (St. Jos. '19) in general practice at Lexington, Mo.

DR. L. C. BLACKBURN (K. S. C. '33), who has been in the service of the U. S. Bureau of Animal Industry, has resigned to take over the practice of Dr. N. E. Gubser (Chi. '11), at Earlham, Iowa.

DR. H. W. UNDERWOOD (Cin. '16), of Clearfield, Pa., has been on the sick list since September 15, 1937, and was in the hospital until December 23. He is staying in Phillipsburg, Pa., until he is able to resume his duties.

DR. F. A. HALL (Corn. '23), of Garrett, Ind., was reëlected as a director and secretary of the Garrett Chamber of Commerce at the annual meeting in February. Dr. Hall is also chairman of the Committee on Conventions.

DR. C. R. PASTORS (O. S. U. '31) resigned from the U. S. Bureau of Animal Industry in February and has reopened the small-animal hospital at 113 South Augusta Street, Staunton, Va., formerly occupied by the late Dr. S. C. Neff.

DR. T. W. WHITE (San Fran. '12), former Director of the Idaho Bureau of Animal Industry, has located at Gooding, Idaho, where he has taken over the practice of Dr. E. H. Ikard (K. S. C. '19), who is now with the U. S. Bureau of Animal Industry.

DR. CHAS. M. SAUNDERS (Wash. '35) terminated his duties with the CCC, at Fort Missoula, Mont., the latter part of January, and has accepted the position of County Veterinarian with the Pierce County Health Department, with headquarters at Tacoma, Wash.

DR. GEORGE W. GILLIE (O. S. U. '07), of Fort Wayne, Ind., has announced his candidacy for the Republican nomination for Congress from the Fourth District at the primary election scheduled for May 3. He served as sheriff of Allen County for four terms.

DR. CARL C. TUCKER (O. S. U. '35), son of Dr. F. C. Tucker (McK. '09), of Claypool, Ind., after nearly three years of general practice at Warsaw, Ind., has entered the commercial field and will be the Ohio representative of the National Laboratories Corporation, with headquarters at Washington Court House, Ohio.

DR. EBER M. MASSINGER (N. Y. C. V. S. '91), of Phoenixville, Pa., recently returned home after a well-earned vacation that took him all the way to the Pacific Coast *via* the Panama Canal. He was obliged to return east by rail, on account of the shipping strike in California, and paid his respects at the JOURNAL office during a short stopover in Chicago.

DR. LEE M. RODERICK (O. S. U. '15), who has been professor of veterinary science at North Dakota State College since 1916, has been appointed professor and head of the Department of Pathology, Division of Veterinary Medicine, Kansas State College. He will fill the place made vacant by the death of Dr. H. F. Lienhardt, in November last. Dr. Roderick will take up his new duties about June. 1.